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PYOGENIC INFECTIVE DISEASES OF THE BRAIN AND SPINAL CORD

MENINGITIS

ABSCESS OF BRAIN

INFECTIVE SINUS THROMBOSIS

BY

WILLIAM MACEWEN, M.D.

GLASGOW

JAMES MACLEHOSE AND SONS

Publishers to the University

1893

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PREFACE.

PYOGENIC infective diseases of the cerebro-spinal system have only of late years begun to be duly appreciated, and though additions are being rapidly made to our experience, the knowledge possessed concerning them is still imperfect. Ten years ago a physician reported in the *Lancet* a case of an abscess of the brain discovered post-mortem in one of the London hospitals, and quoted the remark of an eminent surgeon who saw it, that an abscess of the brain was one of those cases which occurred but once in the course of a lifetime. An individual experience of the last ten years given in this volume, and supported by numerous cases recorded by other surgeons, affords quite a different estimate. Abscess of the brain and other pyogenic intracranial affections occur much more frequently than is usually supposed, and the correctness of this assertion will be more apparent as the knowledge concerning these diseases is increased, and becomes more widely distributed.

Though not sharing the hopelessness of the opinion expressed in 1883 by a distinguished neurologist as to the inutility of operations on the brain undertaken for abscess, the author was then inclined to take a more sombre view of the prospects of recovery from such operations than his subsequent experience has proven to be necessary. He now regards an uncomplicated cerebral abscess, early recognized, accurately localized, and promptly operated on, as one of the most satisfactory of all intracranial lesions, the patient being at once relieved from a perilous condition, and usually restored to sound health. On the other hand, when the more serious lesions of infective meningitis and sinus thrombosis are allowed to advance uninterruptedly beyond a certain limit, the first rapidly involves

the whole cerebro-spinal axis, whose function it benumbs, while the other no less insidiously embraces the general system in a toxic grasp which surgical aid is powerless to unloose. Yet in both of these otherwise fatal affections, early recognition, followed by operation, have in certain cases checked the disease, and as our experience ripens concerning them, the results will doubtless be improved.

Since the majority of pyogenic affections of the brain arise from neglected otitis media, they ought to be regarded as preventable diseases, and their prophylaxis scrupulously attended to.

The author had hoped to add a chapter on the literature of the subject, for which material has been collected, but meanwhile its publication must be deferred. The cases reported in this volume as it stands, and the conclusions drawn therefrom, have been principally founded on personal experience. The points advanced by the author in his address on 'The Surgery of the Brain and Spinal Cord,' in 1888, as diagnosticating large abscess in the temporo-sphenoidal lobe, have been fully borne out in several subsequent cases.

The thanks of the author are due to Dr. Barr for numerous pyogenic infective lesions sent by him, and for the loan of literary and pathological material; to Drs. Forrest and Erskine for several valuable specimens; to Dr. G. Ritchie Thomson for the bacteriological investigations undertaken at the author's request, and for his careful scrutiny of the proofs.

Glasgow, October, 1893.

CONTENTS.

CHAPTER I.—SURGICAL ANATOMY.

	PAGE
Temporal bone as a whole	1
Development ossification	2
External auditory osseous meatus	2
Tympanum	3
Arterial and venous supply of tympanum	5
Communications with tympanum	5
Mastoid antrum	7
Petro-squamosal suture	8
Masto-squamosal suture	8
Supra-meatal triangle	9
Changes in relation to age	11
Eustachian tube	12
Mastoid process and cells	12
Petrous portion	13
Carotid canal	13
Fossa subarcuata	14
The facial canal (aqueductus Fallopii)	14
Its relation to antro-tympanal passage	15
Sigmoid groove	16
Its relation to outer surface of skull	16
Relations of temporo-sphenoidal lobe to temporal bone	17
Relations of cerebellum to posterior fossa	18
Nerves passing through temporal bone	18
Acoustic	18
Facial	18
Superficial petrosal nerves	19

PAGE

Temporal bone—nerves passing through—
Chorda tympani - - - - - - - - - 19
Auricular branch of vagus - - - - - - - 19
Tympanic branch of glosso-pharyngeal - - - - 19
Tympanic plexus - - - - - - - - - 19
Paralysis of facial nerve - - - - - - - 19
Functions of chorda tympani - - - - - - 20
Semicircular canals - - - - - - - - - 21
Vertigo, nystagmus, from irritation of - - - - 21
Perverted and defective ocular and auditory impressions - 21
Frontal sinuses - - - - - - - - - - - 22
The venous supply of head - - - - - - - - - 23
Cerebral veins - - - - - - - - - 23
Superior longitudinal sinus - - - - - - - 24
Sigmoid sinus - - - - - - - - - - 24
Mastoid vein - - - - - - - - - - 25
Posterior condylar vein - - - - - - - - 26
Anterior condylar veins - - - - - - - - 27
Relation of sigmoid sinus to cerebellum - - - - - 28
Cavernous sinus - - - - - - - - - 29
Other sinuses - - - - - - - - - - 30
Diploic veins - - - - - - - - - - 30
Persistent petro-squamosal sinus - - - - - - 32
Obstruction of the sigmoid and internal jugular and its effects 32
Structures which pass through jugular foramen - - - 33
Relation of nerves and artery to cavernous sinus - - - 33
Pterygoid plexus, its intracranial and superficial connections - 33
Mechanism preventing aspiration of the sinuses - - - 33
Intracranial venous arrangement - - - - - 35
Dynamics of sinuses - - - - - - - 37
Lymphatics of head—extra- and intra-cranial - - 38
In relation to their glands - - - - - - 39
Cerebral membranes - - - - - - - - 40
Dura mater - - - - - - - - - 40
Subdural space - - - - - - - - 42
Arachnoid mater - - - - - - - - 43
Arachnoid and subarachnoid space - - - - - 44
Arachnoid villi (Glandulæ Pacchionii) - - - - 45
Cerebro-spinal fluid - - - - - - - 46

PAGE

Cerebral membranes—
Water-bed - - - - - - - - - - 46
Pia mater - - - - - - - - - - 47
Nerve supply to cerebral membranes - - - - - 48

CHAPTER II.—PATHOLOGY OF CEREBRAL ABSCESS AND MENINGITIS.

1. The close relations of meningitis and brain abscess - - - - 49
2. The various intracranial lesions which may result from the pathogenic and saprophytic causes - - - - - - - 49
3. The channels through which the various organisms pass from the outside to the interior of the skull and its contents - - 51
4. Micro-organisms the chief cause of the intracranial pathogenic processes - - - - - - - - - - - 52
5. Kind of injuries and pathological conditions leading to intracranial diseases - - - - - - - - - - - 56
Infective injuries to the skull—punctured wounds penetrating into bone - - - - - - - - - - 56
Punctured fractures - - - - - - - - - 57
Case I. - - - - - - - - - - - 57
Compound fractures - - - - - - - - - 57
Case II. - - - - - - - - - - - 58
Case III. - - - - - - - - - - - 60
Injuries to skull without fractures - - - - - 61
Necrosis from external injury - - - - - - 61
Pathogenic affections of face and scalp - - - - - 61
6. Inflammation of middle ear, relatively to intracranial inflammation 62
Acute inflammation of middle ear may, though rarely, spread into interior of skull - - - - - - - - 62
Otitis media purulenta may exist without rupture of membrana tympani, although accompanied by extensive osseous erosion - - - - - - - - - - - 62
Chronic inflammation of middle ear - - - - - 63
Effects of otitis media—osseous erosion or sclerosis - - 63
Relative frequency of various parts of mastoid to be affected with caries or necrosis - - - - - - - - 64
Peculiarities of anatomical arrangement in temporal bone of young children in relation to disease - - - - 64
Sub-periosteal mastoid abscess - - - - - - 64

PAGE

6. Inflammation of middle ear—

Acute periostitis of mastoid and squamous, passing on to abscess, uncommon - - - - - - - - - 6

Appearances in acute sub-periosteal squamo-mastoid abscess - - - - - - - - - - 65

Cases IV., V., VI. - - - - - - - - - - 66

Mode of infective extension from tympanum - - - - 67

Most frequent seats of osseous perforation—tegmen and sigmoid groove - - - - - - - - - 68

Thrombosis of minute tympanic and antral veins precedes erosion - - - - - - - - - - 68

Dark green discoloration of sigmoid groove, etc. - - 68

Some consequences of osseous erosion and necrosis - - - 68

Formation of extradural abscess - - - - - 69

Case VII. - - - - - - - - - - 69

Case VIII. - - - - - - - - - - 70

Case IX. - - - - - - - - - - 71

7. Mode of infective involvement of membranes and brain - - - 71

Involvement of middle cranial fossa - - - - - 71

Behaviour of dura mater when attacked by infective inflammation from without - - - - - 72

Mode in which inner membranes become affected - - 72

Arteries of brain may become seats of infective embolism or thrombosis - - - - - - 72

How the cerebellar fossa is attacked - - - - - 72

Extradural abscess and sigmoid sinus thrombosis - - 73

Formation of cerebellar abscess - - - - - 74

Formation of cerebellar lepto-meningitis - - - - 74

Dangers of indiscriminate removal of granulation tissue from middle ear - - - - - - - - - 74

8. Subdural abscess—ulceration of brain - - - - - - 75

Case X. - - - - - - - - - - 75

Case XI. - - - - - - - - - - 77

Case XII. - - - - - - - - - - 78

Case XIII. - - - - - - - - - - 79

9. Formation of abscess in middle cranial fossa - - - - - 80

Reasons why right intracranial lesions from otitis media may be more frequent than left - - - - - - 81

Cerebellar and cerebral abscesses may empty themselves through the osseous cranial erosions - - - - 82

PAGE

9. Formation of abscess in middle cranial fossa—
Addenda to mastoid infective lesions - - - - - 84
Tubercle middle ear - - - - - - - 84
Infective disease in infants - - - - - - 84
Extension of mastoid necrosis - - - - - - 84
Extension of middle ear inflammation to Eustachian tube and formation of retro-pharyngeal abscess - 84
Acute cerebro-spinal lepto-meningitis - - - - - 84
Acute serous lepto-meningitis - - - - - - 85
Appearances of serous lepto-meningitis—
During life - - - - - - - - - - 85
Reference to case of acute serous lepto-meningitis, probably tubercular - - - - - - - 86
After death - - - - - - - - - - 86
Acute purulent cerebro-spinal lepto-meningitis - - - 87
Case XIV. - - - - - - - - - - 88
Case XV. - - - - - - - - - - 92
Case XVI. - - - - - - - - - - 96
Case XVII. - - - - - - - - - - 98
Case XVIII. - - - - - - - - - - 100
Purulent encephalitis - - - - - - - - 102
Red softening - - - - - - - - - 102
Case XIX. - - - - - - - - - - 102
White softening - - - - - - - - - 105
Pathogenic embolism - - - - - - - 105
Anæmic and hæmorrhagic necrosis of brain - - - 105
Character of cerebral abscess pus - - - - - - 105
Character of brain tissue in immediate vicinity of abscess and zone beyond - - - - - - - - - 106
Encapsulation of abscess - - - - - - - 107
Description of encapsuled abscess removed from patient - - 108
Calcification of abscess - - - - - - - - 109
Cerebral abscess may become absorbed - - - - - 109
Cerebral abscess after encapsulation may increase from within 109
Cerebral abscess may empty itself externally by pathological processes (Otorrhœa Cerebralis) - - - - - 110
Cerebral abscess may burst or leak into subdural tissue or ventricles - - - - - - - - - - 111
Can an abscess of brain remain *in situ* while the middle ear disease which gave rise to it becomes cured ? - - - 111

PAGE

9. Formation of abscess in middle cranial fossa—
A fresh abscess may form in the periphery of the encapsulated one - - - - - - - - - - - - 112
Size of cerebral abscess cannot be estimated by degree of pressure produced by abscess - - - - - - 112
Size of cerebral and cerebellar abscess - - - - - 113
Hæmorrhage into abscess - - - - - - - 114
Multiple abscess - - - - - - - - - 114
Lowering of temperature and slowing of pulse rate in abscess 114
Necrosis of portion of cerebral tissue - - - - - 115
Case XX. - - - - - - - - - - - 116
Pyæmic abscess of brain - - - - - - - 121
Multiple abscess - - - - - - - - - 122
Parts of brain most apt to be affected with abscess - - 122
Mode of healing of a cerebral abscess after evacuation - - 123
Tuberculosis and carcinosis of the middle ear - - - - - 124
Tubercle of middle ear - - - - - - - - - - 124
Case XXI. - - - - - - - - - - 126
Carcinoma of middle ear and mastoid process - - - - - 128
Treatment - - - - - - - - - - 129
Cases XXII., XXIII., XXIV. - - - - - - 129

CHAPTER III.—SYMPTOMS OF ABSCESS OF BRAIN AND MENINGITIS.

PAGE

Course of abscess of brain divided into three stages - - - - 130
Initiatory stage, symptoms of - - - - - - - - 130
Pain - - - - - - - - - - - 131
Vomiting - - - - - - - - - - 131
Rigors - - - - - - - - - - - 131
Temperature - - - - - - - - - - 132
Cessation of pus from middle ear - - - - - - 132
Duration of initial stage - - - - - - - - 132
Second stage - - - - - - - - - - - 132
Pain deadened—elicited on percussion - - - - 132-133
Slow cerebration - - - - - - - - - 133
Want of sustained attention - - - - - - - 133
Diminution of ability to apply strength - - - - - 134
Tendency to doze - - - - - - - - - 134

PAGE

Second stage—
Temperature, apyrexia - - - - - - - - - 134
Pulse, slow - - - - - - - - - - - 135
Respiration, slow - - - - - - - - - - 137
Constipation - - - - - - - - - - 138
Retention of urine - - - - - - - - - 138
Anorexia the rule - - - - - - - - - 138
Vomiting unusual - - - - - - - - - 138
Convulsions infrequent - - - - - - - - 138
Paralysis - - - - - - - - - - 139
Colour of face not distinctive - - - - - - - 139
Odour from breath - - - - - - - - - 140
Rigors unusual - - - - - - - - - 140
Emaciation - - - - - - - - - - 140
Reflexes uncertain - - - - - - - - - 141
Muscular fibrillæ - - - - - - - - - 141
Optic neuritis - - - - - - - - - - 141
Optic neuritis in otitis media - - - - - - - 142
Aural examination, infective otorrhœa - - - - - 142
Swelling and redness of mastoid (seldom seen) - - - 143
Terminal stage - - - - - - - - - - - 143
Stupor and coma - - - - - - - - - 143
Leakage into membranes or into ventricles - - - - 143
Symptoms of acute lepto-meningitis, - - - - 143
Bursting into ventricles or into membranes - - - - 144
Pyrexia, convulsions, coma - - - - - - 144
Encapsulated abscess may remain at rest for long periods - 144
Abscesses may become absorbed - - - - - - 144
Abscesses may evacuate themselves - - - - - 144
Localizing symptoms - - - - - - - - 145
General pupillary phenomena - - - - - - - 145
Elicitation of differential cranial percussion note - - - - 146
Percussion note in over-distended lateral ventricles - - 148
Percussion note in extensive fractures of skull - - - 149
Value of percussion note in diagnosis of cerebellar lesions - 149
The "latent period" in abscess formation - - - - 149
Evidence of intracranial abscess from quantity of otorrhœal discharge - - - - - - - - - - 150
Evidence from auriscopic examination - - - - - 150

PAGE

Abscess of brain—

Symptoms of empyema of frontal sinuses - - - - 151
Localizing symptoms of abscess in temporo-sphenoidal lobe - 151
None when small - - - - - - - - 151
When large, hemiparesis or hemiplegia - - - - 151
Sequence of hemiplegia - - - - - - - 152
Absence of sensory paralysis - - - - - 152, 158
Cortical facial paralysis - - - - - - - 153
Aphasia - - - - - - - - - - 153
Paralysis of third nerve - - - - - - - 154
Summary - - - - - - - - - 154
Somnambulistic state - - - - - - - 154
Word deafness and psychical blindness - - - - 155
Case XXV. - - - - - - - - - - 155
Case XXVI. - - - - - - - - - - 162
Case XXVII. - - - - - - - - - - 168
Case XXVIII. - - - - - - - - - 171
Case XXIX. - - - - - - - - - - 174
Case XXX. - - - - - - - - - - 177
Case XXXI. - - - - - - - - - - 181
Case XXXII. - - - - - - - - - 182
Localizing symptoms of abscess in frontal lobes - - - 183
Pupillary manifestations - - - - - - - 183
Transient aphasia, - - - - - - - - 183
Case XXXIII. - - - - - - - - - 183
Case XXXIV. - - - - - - - - - 189
Parietal region - - - - - - - - - 193
Motor phenomena - - - - - - - - 193
Case XXXV. - - - - - - - - - - 193
Occipital lobes - - - - - - - - - 194
Symptoms of abscess of cerebellum - - - - - - 194
When small, few localizing - - - - - - 194
When large, slow feeble pulse and respiration - - - 194
Low temperature - - - - - - - - 194
Yawning - - - - - - - - - - 195
Trismus - - - - - - - - - - 195
Syllabic speech - - - - - - - - 195
Optic neuritis, blindness - - - - - - - 195
Brachio-crural paralysis - - - - - - - 195

PAGE

Symptoms of abscess of cerebellum—
Bilateral paralysis - - - - - - - - - - 195
Vertigo and vomiting, marked - - - - - - - 195
Abscess of pons - - - - - - - - - - 195
Alternate hemiplegia - - - - - - - - - 195
Incoordination - - - - - - - - - - 195
Stoppage of respiration, heart beat continuing - - - 196
Cheyne-Stokes respiration - - - - - - - - 196
Signs of distension of ventricles - - - - - - 196
Case XXXVI. - - - - - - - - - - 197
Case XXXVII. - - - - - - - - - - 203
Case XXXVIII. - - - - - - - - - - 207
Case XXXIX. - - - - - - - - - - 209
Case XL. - - - - - - - - - - - 210
Case XLI. - - - - - - - - - - - 212
Case XLII. - - - - - - - - - - - 214
Case XLIII. - - - - - - - - - - - 216
Diagnosis - - - - - - - - - - - - - 221
Differentiation between encephalitis and abscess - - - 221
Differentiation between lepto-meningitis and abscess - - 221
Differentiation between infective thrombosis of sigmoid sinus and abscess - - - - - - - - - - 222
Differentiation between infective systemic poisoning and abscess 223
Differentiation between tumour and abscess - - - - 223
Prognosis - - - - - - - - - - - - - 223
With operation - - - - - - - - - - 223
Without operation - - - - - - - - - - 224
Danger of encysted abscess - - - - - - - - 224
Course of cerebral abscess - - - - - - - - - - 225

CHAPTER IV.—THROMBOSIS OF THE INTRACRANIAL SINUSES.

Introductory note - - - - - - - - - - - - 226
Two forms of thrombosis - - - - - - - - - - 227
Marasmic thrombosis - - - - - - - - - - - 227
Location and ætiology - - - - - - - - - - 227
Pathology - - - - - - - - - - - - 228

PAGE

Marasmic thrombosis—
Symptoms - - - - - - - - - - 229
Diagnosis - - - - - - - - - - 230
Infective or inflammatory thrombosis - - - - - - - 231
Location and ætiology - - - - - - - - - 231
Pathology - - - - - - - - - - 232
Changes in vessel wall - - - - - - - 232
Formation of blood clot - - - - - - - 232
Disintegration of clot - - - - - - - 232
Systemic infection - - - - - - - - 232
Extension to internal jugular - - - - - - 233
Extension to neighbouring venous channels - - - 233
Effect upon neighbouring tissues - - - - - 233
Formation of extracranial abscess - - - - - 233
Lepto-meningitis as complication - - - - - 234
Effects upon the bone and soft tissues - - - - - 234
Systemic infection—pulmonary infarctions - - - - 235
Formative obliteration of sinus possible - - - - - 235
Summary of differences between marasmic and infective thrombosis - - - - - - - - - - 236
Symptoms - - - - - - - - - - 236
General symptoms - - - - - - - - 236
Cephalalgia - - - - - - - - - 236
Vomiting - - - - - - - - - 237
Pyrexia, remittent - - - - - - - 237
Rapid small pulse - - - - - - - 237
Repeated rigors - - - - - - - - 237
Three groups according to dominant symptoms - - - 238
Pulmonary type - - - - - - - - 238
Typhoid type - - - - - - - - - 240
Meningeal type - - - - - - - - 241
Types usually mixed - - - - - - - 242
Complicated with cerebral abscess - - - - - 242
Thrombosis of special sinuses - - - - - - - - 242
Local lesions may excite thrombosis in any of the sinuses - 242
Superior longitudinal sinus thrombosis - - - - - 242
Marasmic and infective causes - - - - - - 242
Symptoms - - - - - - - - - 243
Case XLIV.—Marasmic thrombosis of longitudinal sinus - 243

PAGE

Thrombosis of special sinuses—
Case XLV.—Thrombosis of longitudinal sinus from cario-necrosis of skull - - - - - - - - - 244
Case XLVI.—Thrombosis of longitudinal sinus from infective traumatism - - - - - - - - - - 244
Case XLVII.—Thrombosis of longitudinal sinus from traumatism - - - - - - - - - - - 244
Cavernous sinus thrombosis - - - - - - - - - - 245
Ætiology - - - - - - - - - - - - 245
Infective inflammation in orbital cavity - - - - 246
Infective inflammation in face - - - - - - 246
Infective inflammation in buccal, nasal, and oral cavities and pharynx - - - - - - - - - 246
Infective basal meningitis from caries of sphenoid - - 246
Infective inflammation of cavities leading to nasal fossæ - 246
Infective inflammation of maxillæ - - - - - 246
Symptoms and appearances - - - - - - - 247
Unilateral, bilateral, and alternate - - - - - 247
Pain - - - - - - - - - - - - 247
Exophthalmos - - - - - - - - - 247
Œdema of eyelids and chemosis - - - - - 249
"Choked disc," - - - - - - - - - 249
Implication of pterygoid and pharyngeal vessels - - 249
Œdema of side of face - - - - - - - 249
Enlargement of frontal and facial veins - - - - 250
Nerve symptoms - - - - - - - - - 250
Ptosis, myosis, mydrasis, strabismus, ophthalmoplegia - 250
Alternate involvement of eyeballs - - - - - 250
Duration of the disease - - - - - - - - 251
Case XLVIII.—Cavernous sinus thrombosis due to tumour of base of skull - - - - - - - - - - 251
Case XLXIX.—Cavernous sinus thrombosis due to pustule in nostril - - - - - - - - - - - 253
Case L.—Cavernous sinus thrombosis due to traumatism of orbit - - - - - - - - - - - 254
Case LI.—Cavernous sinus thrombosis due to infective ozena - 254
Case LII.—Cavernous sinus thrombosis due to infective otitis media - - - - - - - - - - - 255
Sigmoid sinus thrombosis - - - - - - - - - - 260
Seldom in infancy - - - - - - - - - - 260

PAGE

Sigmoid sinus thrombosis—
Ætiology - - - - - - - - - - - 260
Extension of infective inflammation from middle ear - 261
Symptons and appearances - - - - - - - 262
Enlargement of glands - - - - - - - 262
Distension of extracranial veins - - - - - 263
Œdema of mastoid region - - - - - - 263
Pain on percussion of the mastoid - - - - - 264
Phlebitis of veins leading from sinus - - - - 264
Pain on pressure along course of jugular - - - - 265
Pain on pressure in upper third of jugular - - - 265
Pain on pressure in posterior cervical triangle - - 265
Enlargement of cervical glands and soft tissues - - 265
Cervical abscess - - - - - - - - - 265
Systemic involvement - - - - - - - - 266
Pulmonary invasion - - - - - - - - 266
Infective enteritis - - - - - - - - - 266
Considerable involvement of sigmoid without marked symptoms - - - - - - - - - - - 267
Differential diagnosis between sigmoid thrombosis, abscess of brain, and meningitis - - - - - - - - 267
Meningitis frequently accompanies advanced thrombosis - 268
Thrombosis of sigmoid sinus, with small cerebral or cerebellar abscess - - - - - - - - - - - 268
Prognosis (general) - - - - - - - - - 268
Marasmic - - - - - - - - - - 268
Infective - - - - - - - - - - 268
Case LIII.—Sigmoid sinus thrombosis from acute affection of throat - - - - - - - - - - - 269
Case LIV.—Sigmoid sinus thrombosis from otitis media - - 271
Case LV.—Sigmoid sinus thrombosis from otitis media - - 275
Case LVI.—Sigmoid sinus thrombosis from otitis media - 278
Case LVII.—Sigmoid sinus thrombosis from otitis media - 282
Case LVIII.—Sigmoid sinus thrombosis from otitis media - 285
Case LXIX.—Abscess under deep cervical fascia resulting from intracranial suppuration - - - - - - 286
Case LX.—Abscess under deep cervical fascia from mastoid suppuration and perforation into digastric fossa - - 287
Cases LXI., LXII.—Empyema of frontal sinus with perforation into orbital cavity - - - - - - - - 287

PAGE

Intracranial invasion from empyema of frontal sinuses—

Case LXIII.—Intracranial invasion from empyema of frontal sinuses - - - - - - - - - - - - 288

Case LXIV.—Intracranial infective invasion from trauma without visible wound - - - - - - - - - 289

CHAPTER V.—TREATMENT.

Prophylaxis against infective intracranial lesions - - - - 291

Rendering scalp wounds and compound fractures of skull aseptic - 291

Rendering wounds of dura mater and brain aseptic - - - - 292

Rendering orbital nasal cavities, and maxillary and frontal sinuses aseptic - - - - - - - - - - - - - - - 292

Treatment of infective postules about nose, face, and lips - - - 293

Treatment of infective wounds of orbital cavity - - - - - 293

Acute purulent otitis media in relation to intracranial affections - - 293

Chronic purulent otitis media in relation to intracranial lesions - - 293

Treatment of otitis media—too frequently neglected - - - - 293

Indications for opening mastoid antrum - - - - - - - 295

Opening of mastoid antrum - - - - - - - - - - 295

Preparation of patient - - - - - - - - - 295

Instruments required - - - - - - - - - 296

Steps of operation - - - - - - - - - - 296

Three points to be noted in conformation of bone—anatomical land marks - - - - - - - - - - 297

Supra-meatal triangle, etc. - - - - - - - - 297

Depth of mastoid antrum from surface of skull and some peculiarities regarding its relative position - - - - - 299

Parts to be noted after antrum is exposed - - - - 300

Exposure of mastoid cells - - - - - - - - - - 301

Opening the tympanic attic - - - - - - - - - - 301

Removal of the ossicles from middle ear - - - - - - 302

Its effect upon hearing - - - - - - - - - 302

Destruction of chorda tympani and its results - - - - - 302

Means of facilitating the closure of the gap in mastoid antrum - - 302

Choice of instruments for opening the mastoid antrum - - - 303

Objections to use of chisel and mallet - - - - - 303

Accidental wounding of sigmoid sinus - - - - - 303

Accidental opening into cerebellum - - - - - 304

Possible dissemination of contents of brain abscess - - 304

PAGE

Choice of instruments for opening the mastoid antrum—
Other instruments—Russian perforator - - - - - 304
Trephine not recommended - - - - - - - 305
Cautions regarding injections of fluid from antrum into middle ear - 305
Dressing antrum and middle ear - - - - - - - - 306
Objects aimed at during wound healing - - - - - - 306
Filling space with avascular connective-tissue barrier - - 306
Establishing permanent opening through antrum, between middle ear and back of external auditory meatus - - 306
Treatment of infective sinus thrombosis - - - - - - 307
How to expose the sigmoid sinus - - - - - - - - 307
Surgical anatomy of - - - - - - - - 307
Extension of operation upon antrum into sigmoid groove - 308
Some pathological conditions which may require attention during operation - - - - - - - - 309
Obliteration of sigmoid sinus - - - - - - - - 309
How to occlude the sinus in case of hæmorrhage - - - - - 309
Admission of air into sinuses - - - - - - - - 310
Ligation of internal jugular vein in thrombosis of sinus - - - 310
Reasons for and against - - - - - - - - 311
Anæsthetics in inflammatory cerebral affections - - - - - 312
Infective meningitis, extradural abscess and abscess of brain - - 312
Infective meningitis and extradural abscess above tegmen tympani and antri - - - - - - - - 312
Caution in injecting fluid through apertures in tegmen - - 313
Ulceration of brain - - - - - - - - 313
Cerebral abscess in temporo-sphenoidal lobe - - - - - 313
Opening through tegmen - - - - - - - 313
Choice of position of opening through external aspects of skull 314
Mode of operating for temporo-sphenoidal abscess - - - 314
Exploratory instruments - - - - - - - 314
The escape of pus and the removal of sloughs - - - - 315
Washing abscess cavity - - - - - - - - 317
Double opening into abscess cavity - - - - - 318
Behaviour of abscess cavity after evacuation - - - - 318
Drainage of abscess cavities - - - - - - - 318
Treatment of external wound - - - - - - - 319
Dressing of external wound - - - - - - - 319
Dressings - - - - - - - - - - 319

PAGE

Cerebellar abscess—
Opening of cerebellar abscess - - - - - - - 320
Opening of frontal abscess - - - - - - - - - 321
Opening of abscess in third frontal convolution - - - - - 321
Exposure of frontal sinuses - - - - - - - - - 322
Treatment of patient after evacuation of brain abscess - - - - 324
Treatment of infective meningitis - - - - - - - 325

CHAPTER VI.—RESULTS.

All but recent cases of intracranial pyogenic infective diseases defectively reported - - - - - - - - - - 326
Recent cases of temporo-sphenoidal abscess - - - - - - 326
Chronological statement of earlier cases of brain abscess under author's care - - - - - - - - - - - - - 326
Résumé of author's results - - - - - - - - - 327
Fifty-four cases of ablation of mastoid antrum and cells - - - 327
Thirty-eight cases in which mastoid antrum and cells only were involved by extension from the tympanum - - 328
Sixteen cases in which petrous also involved - - - - 328
Statistical table of ablations of mastoid antrum and cells for pyogenic otitis media - - - - - - - - - - - - 329
Seventeen cases of infective pachy-meningitis externa, in which operations were performed - - - - - - - - - - 329
Twelve cases of infective purulent lepto-meningitis, of which six were operated on - - - - - - - - - - - - 329
Six cases of cerebro-spinal lepto-meningitis, of which five were operated on - - - - - - - - - - - - 330
Twelve cases of recovery after operation for sigmoid sinus thrombosis - 331
Statistical table of infective intracranial lesions - - - - - 332
Twenty-five cases of abscess of brain, of which nineteen were operated on, and eighteen recovered - - - - - - - - 332
Ten cases of temporo-sphenoidal abscess - - - - - 332
Eight cases of cerebellar abscess - - - - - - 333
Statistical table of intracranial abscess - - - - - - - 333
Twenty-two brain abscesses evacuated, with twenty-one recoveries - 334
Five cases of extradural abscess, which recovered after operation - - 334

ADDENDUM, - - - - - - - - - - - - - - 335
INDEX, - - - - - - - - - - - - - - 339

TEMPERATURE CHARTS.

CHART		PAGE
A.	Purulent cerebro-spinal lepto-meningitis,	90
B.	Purulent cerebro-spinal lepto-meningitis,	94
C.	Abscess of brain,	134
D.	Infective thrombosis of cavernous sinus,	135
E.	Infective purulent cerebro-spinal lepto-meningitis,	136
F.	Abscess of brain,	157
G.	Suppurative lepto-meningitis and recurrent cerebral abscess,	166
H.	Traumatic cerebral abscess,	186
I.	Cerebellar abscess,	199
J.	Suppurative lepto-meningitis,	219
K.	Infective sinus thrombosis,	237
L.	Infective sinus thrombosis,	238
M.	Infective thrombosis of cavernous sinus,	258
N.	Infective sigmoid sinus thrombosis,	273
O.	Infective sigmoid sinus thrombosis,	277
P.	Infective sigmoid sinus thrombosis,	283

LIST OF ILLUSTRATIONS.

FIG.		PAGE
1.	Temporal bone of an infant—external aspect,	2
2.	Temporal bone of an infant—internal aspect,	2
3.	Mastoid antrum and tympanic cavity in infant,	4
4.	Mastoid antrum and tympanic cavity in infant, viewed obliquely from below,	4
5.	Descending plate of left squamous in infant—internal aspect,	6
6.	Descending plate of left squamous in infant—external aspect,	6
7.	Imperfect ossification of tympanic ring,	8
8.	Right temporal bone, male 15 years—masto-squamosal suture,	10
9.	Left temporal bone, male 15 years—masto-squamosal suture,	12
10.	Preparation of right temporal bone showing the course and relations of facial canal,	14
10A.	Course of sigmoid sinus relative to exterior of skull,	16
11.	Left temporal bone—inner aspect. The anterior knee of the sigmoid groove,	18
12.	Left temporal bone—internal aspect. Showing tegmen tympani and petro-squamosal suture,	20
13.	Section of right temporal bone showing relations of mastoid antrum,	26
14.	Section of left temporal bone showing communication between mastoid antrum and middle ear,	26
15.	Section of left temporal bone showing the mastoid antrum and cells,	28
16.	Section of left temporal bone showing mastoid antrum and cells,	28
17.	Anastomosis of cavernous sinus with extracranial veins (from Quain after Hirschfeld and Leveillé),	29
18.	Schema of intra- and extra-cranial venous anastomosis,	31
19.	Schema of sigmoid sinus in relation to jugular bulb,	36
20.	Coronal head section—sigmoid sinus and jugular dome,	36
21.	Section through the right petro-mastoid and portion of the occipital bone, showing height of internal jugular dome in relation to the vertical limb of sigmoid sinus,	38
22.	Section of upper part of brain, subarachnoid space, filled by trabeculæ (from Quain, after Key and Retzius),	43
23.	Section of posterior and inner parts of brain, showing "water-bed" (Quain, from Key and Retzius),	44
24.	Cerebral pia mater,	48

FIG.		PAGE
25.	Acute sub-periosteal mastoid abscess,	64
26.	Acute sub-periosteal squamo-mastoid abscess,	64
27.	Acute sub-periosteal squamo-mastoid abscess,	66
28.	Acute sub-periosteal squamo-mastoid abscess,	66
29.	Schema of infective invasion of membranes,	71
30.	The relations of the base of the temporo-sphenoidal lobe to the tympanic attic,	72
31.	Schema of extradural abscess,	73
32.	Erosion from infective processes in knee of sigmoid groove,	74
33.	Schema of ulceration of brain,	75
34.	Pathological erosion between cerebellar fossa and exterior of mastoid,	76
35.	Infective erosions in knee of sigmoid sinus,	78
36.	Commencing infective invasion of left temporo-sphenoidal lobe,	80
37.	Abscess in left temporo-sphenoidal lobe,	84
38.	Abscess in right temporo-sphenoidal lobe,	88
39.	Cerebral abscess, abscess in left temporo-sphenoidal lobe,	90
40.	Abscess in temporo-sphenoidal lobe (seen in section),	92
41.	Abscess of brain, encysted abscess in right temporo-sphenoidal lobe (seen in section),	94
42.	Cerebellar abscess and purulent encephalitis surrounding ventricles,	96
43.	Encysted cerebellar abscess,	102
44.	Section of encysted cerebellar abscess,	102
45.	Encapsulated abscess,	106
46.	Encapsulated abscess,	106
47.	Necrosis of portion of brain,	114
48.	Necrosis of brain,	116
49.	Necrosis of brain,	118
50.	Schema illustrating sequence of paralysis caused by abscess in temporo-sphenoidal lobe,	152
51.	Schema illustrating production of transient aphasia by an abscess in the white substance of the left third frontal convolution,	191
52.	Position of sigmoid sinus marked by pathological processes on cerebellum,	234
53.	Facial expression in cavernous sinus thrombosis, early stage,	248
54.	Facial expression in cavernous sinus thrombosis, later stage,	248
55.	Facial expression in cavernous sinus thrombosis, facial stage,	249
56.	Infective invasion of the frontal sinus with intra-orbital abscess,	288
57.	Supra-meatal triangle,	298
58.	Surface markings on skull,	308
59.	Surface markings on skull,	310
60.	Instruments,	304

Figures 1-16, 19-21, 25-38, 40, 42-46, 48, 49, 52-59 *are engraved by Mr. J. D. Cooper, London. Figs.* 24, 39, 41, 47, *and* 60 *are engraved by Messrs. T. & R. Annan & Sons, Glasgow, and Figs.* 17, 22, *and* 23 *are reproduced by permission from "Quain's Anatomy." With the exception of Figs.* 17, 22, *and* 23, *all the illustrations in this book have been specially engraved for it from the author's sketches or specimens.*

CHAPTER I.

SURGICAL ANATOMY.

With the view of aiding the reader regarding points which are of importance for the appreciation of the details bearing on the structures involved in pyogenic infective diseases of the brain and its membranes, a short chapter on the parts of the head most concerned is introduced. The constantly recurring references to the temporal bone in the succeeding chapters, the importance of the structures which pass through it, and its relations to the encephalon, are sufficient reasons for the prominence allotted to it in this place.

TEMPORAL BONE.

The temporal bone is one of the most complex, and, in its relations, one of the most important bones of the skull. Besides containing in its labyrinth the beautiful and delicate organ of hearing and that pertaining to equilibration, it is related to and traversed by many nerves. It contains the tympanic plexus, transmits the great motor nerve of the face and the sympathetic: the apical part of its petrous, supports the Gasserian ganglion, from which the motor nerves of the lower jaw and the sensory nerves of the fore part of the cranium and face descend. It forms part of the jugular foramen, through which pass, besides the jugular vein, the glosso-pharyngeal, vagus, and spinal accessory nerves. It not only harbours the petrosal, sigmoid, and a portion of the lateral sinus, while its apex is in immediate contact with the cavernous sinus, but its substance is extensively intersected by a network of minute bloodvessels. The temporal contains within it a series of cavities clothed with mucous

membrane continuous from the pharynx to the mastoid cells. Disease in the interior of the temporal bone, besides being capable of destroying on the affected side the mimetic play of the features, may lead, if it occurs at a sufficiently early period of life, to unilateral imperfect development of the bones, the muscles of the face, and the salivary glands. Not only may pathological changes cause destruction of the organ of hearing and impairment of equilibration, but through destruction of the facial, the organs of vision, taste, and smell may become affected. Nor are these the worst results, as extension of disease from the temporal bone may occasion the most widespread and fatal maladies, implicating the brain, the spinal cord, the lungs, and intestines.

The temporal bone is often regarded too exclusively as belonging to the organ of hearing. Though the acoustic apparatus plays an important rôle in the pathology of this bone under ordinary circumstances, yet there are diseases in the presence of which the organ of hearing, as such, becomes overshadowed, and for the relief of which it may require to be encroached upon or swept away. In dealing with the temporal bone in this work the acoustic apparatus finds no place, any reference made to it, or to any of its component parts, is merely incidental to the comprehension of diseases of a more general kind, or of the paths by which they travel.

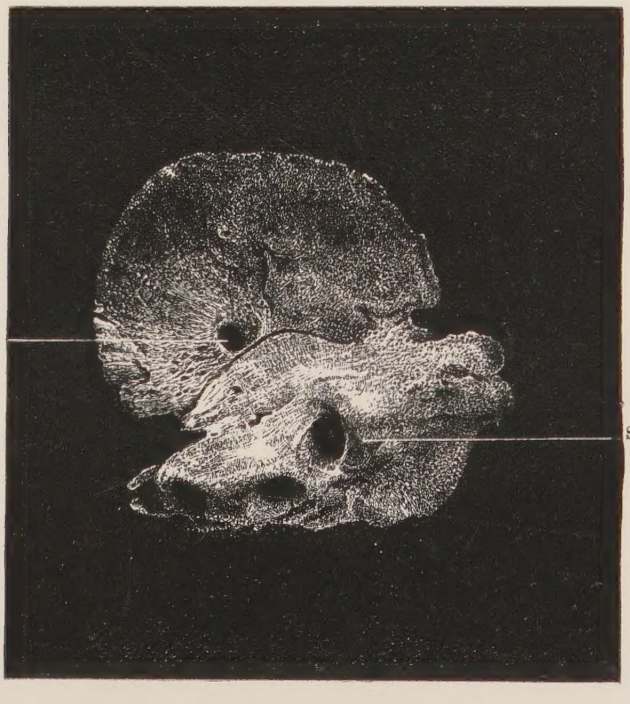
There are two forms of ossification seen in the temporal. The squamosal and tympanal are produced from fibro-cartilage, the petrosal and styloid from cartilage. The petrous, including the mastoid, is more prone to inflammatory destruction than the squamosal and tympanal. The descending plate of the squamous is however an exception, as it is often involved in inflammatory processes extending from the tympanum. The ossification of the temporal proceeds from eight centres: one for the squamous, two for the styloid, one for the tympanic ring, and four for the petrous. Though these various parts have nearly coalesced about the fifth month of intra-uterine life, yet, during the first years, the points of junction are very vascular, and occasionally are even separable by maceration. This has an important bearing on disease, as the greater vascularity favours the spread of inflammatory processes, and tubercle finds therein a suitable nidus.

External auditory meatus.—The osseous portion of the auditory canal is much shorter in the child than in the adult. At birth,

FIG. 1.—TEMPORAL BONE OF AN INFANT—EXTERNAL ASPECT.

The right temporal bone of an infant ten days old, seen from without, which shows the tympanic ring forming the osseous part of the external auditory canal. A portion of silver wire is inserted into the *aqueductus Fallopii* and issues through the unossified part of the aqueduct into the tympanic cavity. Above the tympanic ring and between it and the glenoid fossa is the foramen for the passage of the *petro-squamosal* sinus, a fœtal structure (which occasionally persists through life), and which empties its blood into the external jugular vein.

FIG. 2.—TEMPORAL BONE OF AN INFANT—INTERNAL ASPECT.



The right temporal bone of an infant ten days old, viewed from within. The subarcuate fossa situated above the internal auditory meatus is very wide. The foramen for the petro-squamosal sinus is also seen on the squamous portion of the bone.

To face page 2.

it is a mere ring, which in after life develops outwards, but the external auditory canal is relatively as long in the child as in the adult, the difference being made up in the cartilaginous portion. In the adult, the osseous portion forms two-thirds of the total length of the external auditory canal. Hence the mastoid antrum and middle ear are nearer the surface of the skull in the child than in the adult. In the adult, the posterior wall of the external osseous canal slopes from behind forward and inward. A perpendicular line drawn through the centre of the osseous outlet of the external auditory meatus will in the majority of cases correspond to the plane of the posterior wall of the tympanic cavity. In line with the external osseous meatus lies the horizontal portion of the carotid canal in its course through the temporal bone.[1] An instrument introduced in that line penetrating the inner wall of the tympanum from one-sixteenth to one-eighth of an inch would open the carotid canal.

In the infant.—The tympanic ring throws out two small tubercles, one before, the other behind; these, meeting in the floor of the meatus at the posterior portion of the glenoid fossa, form a foramen which becomes occluded about six years of age, but which occasionally remains permanently patent (see Fig. 7), or may be covered by a very thin plate. This aperture, connected with the tympanic cavity, is situated over the part of the parotid gland which lies in the posterior portion of the glenoid fossa. In the infant, the membrana tympani is placed obliquely but nearly horizontal, while in the adult, it is much nearer the upright position.

The tympanum[2] is an irregularly shaped cavity continuous with the Eustachian tube in front and the mastoid antrum and cells behind. Its long axis is antero-posterior, in which it measures half an inch (13 mm.); its breadth is greater above than below, the former being about three-sixteenths of an inch (4 mm.). Its outer wall is partially osseous, but consists chiefly of the membrana tympani. When the tympanic cavity is exposed for the removal of pathological products by making an opening into it through the antrum, four prominent features may be recognized on its inner wall. First, the promontory caused by the first turn of the cochlea; second, the fenestra ovalis, covered by the stapes (which may in disease

[1] See Atlas of Head Sections, by William Macewen, M.D. (Glasgow, MacLehose, 1893), Plates 39 and 46.

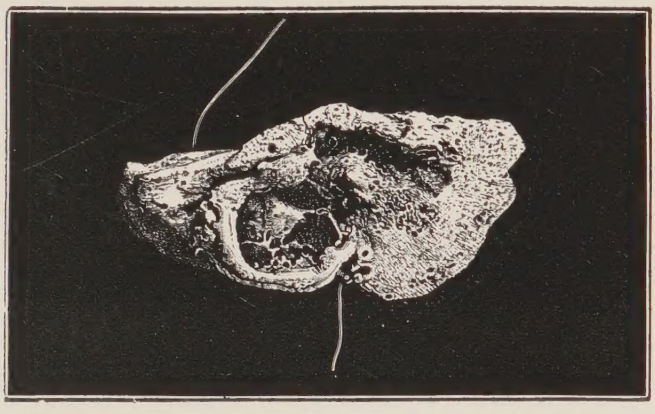
[2] See Atlas of Head Sections, Plates 7, 33, 44, 46.

have disappeared); third, the Fallopian canal (Figs. 1, 3 and 4), lying on its posterior part, between the fenestra ovalis and the roof of the tympanum; and fourth, the fenestra rotunda, with the pyramid between it and the fenestra ovalis, at its posterior and lower part. The floor is very narrow, and is separated by a thin plate of bone from the jugular fossa behind and the carotid in front. The tympanic attic[1]—the posterior and upper part of the tympanum—opens into the mastoid antrum, by an irregularly formed, though somewhat triangular passage, each wall of which measures about 4 mm. There are besides, occasionally, smaller apertures opening directly into the mastoid cells. The anterior extremity of the tympanum is narrow, the roof descending till it meets the osseous part of the Eustachian tube, with which it is continuous. The tegmen tympani is a wide somewhat triangular plate, which not only forms the roof of the tympanum, but also covers in front the Eustachian tube as it makes its exit from the tympanum, and behind, the mastoid antrum. The latter portion is sometimes termed the tegmen antri. The tegmen tympani is bounded on the outside by the petro-squamosal suture (Fig. 12), and on the inside by a groove running outwards and ending in the hiatus Fallopii, or by the eminentia arcuata over the superior semicircular canal. The tegmen is very thin, especially over the mastoid antrum, where it may be translucent or even transparent; and not unfrequently there are defects in this plate, filled in by membrane, upon which the dura mater rests. When this thin plate is fractured, and its closely adhering brain membranes are ruptured, cerebro-spinal fluid escapes into the middle ear. The tegmen tympani is one of the points through which inflammation, arising in the tympanic cavity, spreads to the meninges and brain. Abscesses of the temporo-sphenoidal lobe have been known to discharge themselves at this point, through an opening which probably existed prior to the abscess formation. At the posterior part of the tegmen proprium tympani, there are minute foramina transmitting veins to the superior petrosal and sigmoid sinuses. These veins are increased in size, and probably also in number, by chronic inflammatory action.

The mucous membrane of the tympanum is continuous in front with that of the pharynx, through the Eustachian tube, and behind with the mastoid cells through the mastoid antrum.

[1] See Atlas of Head Sections, Plates 7, 16, 40.

FIG. 3.—MASTOID ANTRUM AND TYMPANIC CAVITY IN INFANT.



The petro-mastoid portion of the left temporal bone of an infant ten days old, from which the squamous with its ascending plate has been removed at the petro-squamosal and masto-squamosal sutures, exposing the mastoid antrum, the Fallopian canal (through which a silver wire passes), and the passage between the tympanum and the mastoid antrum.

FIG. 4.

The same as above, viewed obliquely from below, showing the cancellated tissue in the floor of the mastoid antrum. The silver wire passes through the Fallopian canal, a loop of it projecting over the foramen ovale at a part where the canal is not yet closed by bone.

To face page 4.

Hence diseases of the pharyngeal mucosa may spread by continuity of structure, not only into the tympanum, but also into the remote recesses of the mastoid adjacent to the sigmoid groove.

The chain of ossicles extending from the membrana tympani to the foramen ovale are not infrequently surrounded by masses of granulation tissue, and suffer erosion from pathological processes, for the complete eradication of which the malleus and incus may require removal. In many instances after the removal of these two ossicles and the greater part of the membrana tympani, a considerable degree of hearing is retained.

Arterial and venous supply of the tympanum.—The fore part of the tympanum is supplied by the tympanic branch of the internal maxillary, which passes through the Glaserian fissure; its posterior part and the mastoid cells by the stylo-mastoid branch of the posterior auricular artery which passes through the Fallopian canal. There are also branches from the carotid in its osseous canal, and from the petrosal branch of the middle meningeal, through the hiatus Fallopii. The veins from the tympanum empty into the sigmoid and petrosal sinuses and into the temporo-maxillary vein. (See further under Intracranial Venous Circulation, page 27 *et seq.*)

The tympanic cavity is surrounded by many apertures, through which pathological processes may extend. The following are the principal:—

Communications with tympanic cavity.

1. The external auditory meatus.
2. The Eustachian tube and canal for the tensor tympani muscle.
3. The mastoid antrum and cells.
4. The petro-squamosal fissure running the whole length of the tegmen tympani, open in early years, and marked by numerous channels in after life.
5. The masto-squamosal fissure, open in childhood, occasionally remaining unossified in after life, and marked by numerous foramina when ossified.
6. The fenestræ ovalis and rotunda communicate with the vestibule and cochlea respectively, and the former communicates with the internal meatus; a venous canal from the cochlea communicates with the pyramidal part of the jugular fossa.

7. The Fallopian canal, having communications with the tympanic cavity: in infancy, by a defect in its anterior wall above the fenestra ovalis, and permanently by the aperture for the stapedius muscle and the small nerve twig for the same; by the channel for the chorda tympani, which again pierces the wall of the tympanum at the canal of Huguier in the fissure of Glaser; by an arterial twig—the stylo-mastoid branch of the posterior auricular, which passes into the tympanum.

   The Fallopian aqueduct opens above the cribriform plate into the internal meatus, and through the hiatus Fallopii and a smaller hiatus in front of this (transmitting the greater and lesser superficial petrosal nerves respectively) into the middle cranial fossa, so inflammation may extend along the perineural sheaths and set up lepto-meningitis of both posterior and middle fossæ.
8. The foramen between the carotid canal and the jugular fossa for Jacobson's nerve to the sympathetic plexus and inner wall of the tympanum.
9. The fossa subarcuata—in childhood a wide foramen transmitting a vein to the cells under the superior semicircular canal, and at a still earlier period to the mastoid cells near the outer aspect of the infantile mastoid.
10. The lateral and sigmoid sinus—by means of numerous minute veins through the posterior and lower part of the tegmen tympani and through the mastoid antrum and cells.
11. The superior petrosal sinus—by means of numerous minute venous channels.
12. The inferior petrosal sinus—by means of veins from the labyrinth.
13. The temporo-maxillary vein—by many minute veins from the anterior part of the tympanic cavity.
14. Free communication through the bone with the dura matral system of veins. This may be seen in plates[1] Nos. 19 and 20, where the veins are issuing from the tegmen.
15. The carotid canal—pierced by several foramina for nutrient vessels and branches of the sympathetic nerves to the tympanum, also by small veins.

[1] See Atlas of Head Sections, Plates 19, 20.

FIG. 5.—DESCENDING PLATE OF LEFT SQUAMOUS IN INFANT, INTERNAL ASPECT.

The squamous portion of the left temporal bone from an infant ten days old. It is viewed from within and shows the descending plate of the squamous. (Enlarged.)

FIG. 6.—DESCENDING PLATE OF LEFT SQUAMOUS IN INFANT, EXTERNAL ASPECT.

The squamous portion of the left temporal bone from an infant 10 days old, viewed from without. Showing the descending plate of the squamous. (Enlarged.)

To face page 6.

16. The jugular fossa—the roof of which is pierced by a great number of veins communicating with the floor of the tympanum and the labyrinth, and also by the foramen for the auricular branch of the vagus (Arnold's), communicating with the Fallopian canal in its course.
17. The tympanic branch of the internal maxillary artery which enters at the fissure of Glaser, to supply the fore part of the tympanum.

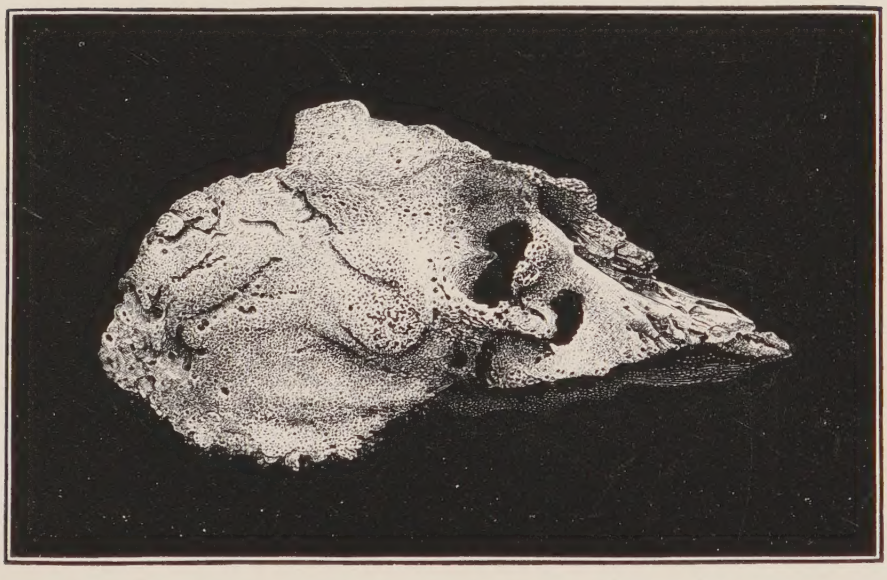
The mastoid antrum[1] is situated behind the tympanic cavity, on a level with, or slightly below, the attic of the tympanum; the Fallopian aqueduct (Fig. 10), which lies on the inner wall of the passage between the antrum and the tympanum, may be taken as the boundary between the two. Like the attic, the antrum varies greatly in size, the tympanic atrium being in this respect much more constant. The antrum approaches nearer the outer surface of the skull, and is separated by a thinner portion of bone from the cranial cavity than is the tympanum. The tegmen antri lies outside of the ridge of the superior semicircular canal. Its floor is frequently on a lower level than the passage by which it communicates with the tympanum, hence drainage of this cavity through the external ear is difficult, and fluid contained therein is apt to gravitate into the cells of the mastoid rather than into the tympanic cavity. Occasionally in childhood the whole mastoid is one large space continuous with the antrum and the middle ear. The anterior wall dividing the antrum from the inner part of the external auditory canal is thin, sometimes defective, and secretion has been known to escape through this wall from the antrum into the external ear. Its external wall consists of the vertical plate of the squamous which extends downwards from the temporal ridge. This plate is marked off from the mastoid by the masto-squamosal suture, which is wide in infancy and occasionally persistent through life (Figs. 8 and 9). The inner wall of the antrum is located at a variable distance from the surface of the skull, the variability depending on the general form and thickness of the cranium and the size of the antrum itself. At the base line of the supra-meatal triangle it varies from nine-sixteenths of an inch in depth to an inch and a quarter—found in one case—the average of its deepest part is three-quarters of an inch.

[1] See Atlas of Head Sections, Plates 9, 17, 23, 25, 27, 33, 34, 35, 40.

In infancy, the squamous with its descending plate is separable after maceration at the masto-squamosal and petro-squamosal sutures. When this is effected, the antrum and tympanum are exposed (Figs. 3, 4, and 5). The petro-squamosal suture is seen on the inner aspect of the temporal, extending from the notch at the lower border of the squamous in front to the notch at its lower border behind (Fig. 12). Along this suture in fœtal life runs the petro-squamosal sinus, extending from the junction of the lateral and sigmoid sinuses to an aperture in front of the squamous near, but behind, the glenoid fossa, through which it passes to join the external jugular vein (Fig 1). Occasionally this sinus persists through life. It existed in a temporal bone taken from a ten days old child, and the foramen for it is seen in Fig. 1. Numerous veins pass from the tympanic cavity to this sinus. After its obliteration, the veins from the tympanum to the lateral and superior petrosal sinuses increase in size, and apparently become more numerous. In children this suture often remains unossified, soft parts alone existing between the dura mater and the tympanic cavity. Through this channel inflammation and pathogenic infection may extend to the sinuses and directly to the cerebral membranes. On the external aspect of the temporal at the junction of the descending plate of the squamous with the mastoid, the *masto-squamosal suture* extends from the notch at the junction of the parietal, squamous, and mastoid, to a position behind the tympanal ring (Figs. 8 and 9). This suture persists till puberty in many instances, and in some remains permanently unossified. Traces of it are found in the shape of foramina and depressions, even after ossification is completed, and through these many minute veins pass from the tympanic cavity to the exterior of the skull. The position of this suture is easily seen on reflecting the periosteum from the mastoid. Besides having been found frequently in children, it has been traced by the author during operation at the ages of 12, 13, 14, 15, 18, and 29 years.[1] As long as this suture remains unossified inflammation may spread, and suppuration may find an outlet from the tympanic cavity to the outside of the skull. This often happens in children, the

[1] Dr. Thomson at the author's suggestion examined 450 temporal bones, and found the masto-squamosal suture present in 67 of the 450, all of which belonged to adults. Kirchner of Wurzburg found the masto-squamosal suture to exist complete on both sides in 5 per cent., and indications of its presence on one or both sides in over 20 per cent. of a series of skulls examined by him. Some skulls of persons aged 68, 70, and 83 years, had this suture distinctly marked.

FIG. 7.—IMPERFECT OSSIFICATION OF TYMPANIC RING.



Portion of right temporal bone from child 2½ years old. The petro-mastoid with the descending portion of the squamous viewed from without, showing the imperfect ossification of the tympanic ring. The mastoid was very vascular, the marking for its numerous vessels are clearly indicated on its surface. Several of the foramina seen on its surface pass directly through the bone to the sigmoid groove, thus uniting the sigmoid sinus with the superficial veins on the external surface of the mastoid. The masto-squamosal suture is still plainly visible.

To face page 8.

inflammation extending from the tympanic cavity forming an abscess over the mastoid.

The position of the masto-squamosal suture is of surgical importance, as it marks the lower and posterior limit of the descending plate of the squamous, which forms the outer wall of the antrum. Operative measures confined to this plate, and not penetrating through it to the underlying petrous bone or to the inner wall of the antrum, are safe from injuring the aqueductus Fallopii on the one hand, and the sigmoid sinus on the other.

The ridge formed by the posterior root of the zygoma indicates by its lower border the level of the roof of the mastoid antrum, and a few lines above is the level of the base of the brain, though these relations may alter in some peculiarly constructed skulls. In two instances the antrum was found nearer the middle line and on a somewhat higher plane than the overhanging outer border of the floor of the middle fossa. The level of the floor of the adult skull at the tegmen antri is on an average less than a quarter of an inch above the roof of the external osseous meatus. In children and adolescents from one-sixteenth to one-eighth of an inch. It is especially thin above the anterior aspect of the roof of the external osseous meatus. The glenoid fossa—which can be made out by passive movement of the lower jaw—also serves as a guide to the level of the base of the brain, which is situated about one-eighth to a quarter of an inch above.[1]

The mastoid antrum may be reached by an opening made at the base of the triangle formed by the posterior root of the zygoma running somewhat horizontally above; the portion of the descending plate of the squamous which forms the arch of the osseous part of the external auditory meatus below, and a base line uniting the two, dropped from the former on a level with the posterior border of the external auditory meatus. At this point there is usually a depression in the bone—though occasionally there is a slight prominence as if the antrum had bulged at that point. The apex of this triangular depressed area points forward. The author proposes to name this area the supra-meatal triangle.[2] Roughly speaking, if the orifice of the external osseous meatus be bisected horizontally, the upper half would be on the level of the mastoid antrum. If this segment be again bisected vertically

[1] Dr. Birmingham gives some interesting details on these measurements. See *Trans. Royal Academy of Medicine of Ireland.* Vol. viii. 1890. p. 250.

[2] See Figs. 8 and 9, and Schema, Fig. 56.

its posterior half would correspond to the junction of the antrum and middle ear, and immediately behind this lies the supra-meatal fossa.

Dr. Thomson was asked to ascertain for the author the presence or absence of the supra-meatal triangle in two hundred and twenty-five skulls. By the kindness of Professors Young and Cleland, the skulls in their collections and in the Hunterian Museum, as well as 25 in author's possession, were carefully inspected with the following result. In 426 of the 450 temporal bones examined the triangle was definite, and of the remaining 24 it was recognizable in 22. It was, therefore, present in 99·5̇ per cent. of all, and well marked in 94·6̇ per cent. Of the 450 temporal bones 250 were British, and in all but 10 of these the triangle was well marked, and in these 10 the boundaries were faintly marked. In 200 foreign temporal bones of all races the triangle was well marked in all but 12, and in only 2 were its boundaries indefinable.

Körner has endeavoured to obtain from the expression of the cranial formation data for determining in the living body the relation of the floor of the middle fossa to the external auditory meatus. Körner's cranial index is obtained by measuring with callipers, first the distance from the bridge of the nose to the most prominent point of the occiput; and, secondly, that of the parietal tuberosities from each other. The first measurement is then divided by the second, the result being a number called the 'cranial index.' In a general way he finds that the middle cranial fossa in dolicocephalic skulls is higher above the external auditory meatus than in those of brachycephalic. With a cranial index 1·55 to 1·40, the floor of the middle cranial fossa lies, according to this author, 11·5 mm. on an average above the external auditory meatus, at the most 17 mm., and at the least 7 mm.; with a cranial index 1·39 to 1·30, the average distance is 8·8 mm., at most 15 mm., and at least 4 mm.; with a cranial index 1·29 to 1·20, the average is 5·1 mm., maximum 9 mm., minimum 2 mm.; with a cranial index 1·19 to 1·07, the average is 4·8 mm., maximum 7 mm., minimum 2 mm. With a cranial index 1·55 to 1·40 the floor of the middle cranial fossa is situated above the spina supra meatum 15·3 mm. on the average, at the most 17 mm., and at the least 10 mm.; with a cranial index 1·39 to 1·30 the average distance is 12·1 mm., at most 17 mm., and at least 7 mm.; with a cranial index 1·29 to 1·20 the average is 7·6 mm., maximum 12 mm., minimum 5 mm.; with a cranial index 1·19 to 1·07, the average is 5·8 mm., maximum 9 mm., and minimum 4 mm.

"Uber die Möglichkeit, einige topographisch wichtige Verhältnisse am Schläfenbeine aus der Form des Schädels zu erkennen."—*Zeitschrift für Ohrenheilkunde*, Bd. xvi. Gruber, "Diseases of the Ear." London. 1890.

When the descending plate of the squamous has been penetrated through the supra-meatal triangle, the antrum is exposed near the passage communicating with the tympanum. A probe ought to be inserted into this passage to mark its position and direction. A probe may likewise be used to explore the size and contour of the

FIG. 8.—RIGHT TEMPORAL BONE, MALE 15 YEARS—MASTO-SQUAMOSAL SUTURE.

Right temporal bone of male, 15 years, showing masto-squamosal suture, dividing the mastoid from the descending plate of squamous.

To face page 10.

antrum, as it is not only a cavity subject to great anatomical variation, but is liable to greater pathological transformation. After the antrum has been opened, further measures may require to be adopted as the disease demands. The Fallopian canal, lying nearer the outer part than the middle of the floor of the antro-tympanal passage, and from this crossing to the inner wall of this passage, must be carefully avoided. This is especially necessary if the disease to be removed affect the tympanic cavity, the attic of which then requires to be opened. This is best done by removing the upper and outer wall of this passage and exposing the upper part of the tympanic cavity—the nearer to the external roof the further from the facial. The posterior recesses of the antrum may be freely exposed as far as its outer covering—the descending plate of the squamous—extends, but the sigmoid groove will be exposed if one penetrates deeply through the mastoid in a backward and inward direction. The tegmen tympani may be easily reached, and, by penetrating through this thin plate, the base of the middle lobe of the cerebrum may be exposed. The tegmen tympani descends forwards and *downwards* from the posterior part of the tegmen covering the antrum, and therefore may be easily penetrated above the anterior part of the tympanum were operative procedures carried forward on the same level as those on the outer part of the upper wall of the antrum. This ought to be borne in mind when the object is to preserve the tegmen, and not to expose the floor of the middle fossa of the skull.

In relation to age.—At birth, the labyrinth, the tympanic cavity and the mastoid antrum are nearly of full size. The triangular plate of the tegmen tympani is then distinct, but it is not ossified to the squamous, as the petro-squamosal suture is filled by membrane, defects in which may expose the tympanum. During early years this suture may remain unossified, and sometimes so persists through life. The masto-squamosal suture at birth is still patent, and there are not infrequently osseous defects in the descending plate of the squamous through which the antrum is exposed. The superior semicircular canal is very prominent in infancy, and has under it the wide fossa subarcuata. The layer of osseous tissue separating the antrum from the sigmoid sinus is, in infancy, generally from 5 to 7 mm. in thickness, though the distance between the lower part of tympanum and sigmoid is often much less. The fœtal tympanum is filled with thick mucous tissue, so that there is no air space. The thickness of the mucous tissue rapidly

diminishes after birth, until in the adult it is very thin, consisting of a few layers of epithelial cells closely covering the bony walls, adherent to the periosteum saving where the vessels and nerves intervene.

The Eustachian tube[1] ascends from the pharynx upwards, backwards, and outwards. Where it is formed of soft parts, the lumen is potential like the urethra. Air passes into the cavity of the tympanum through the Eustachian tube during deglutition. At one part of its course it lies parallel with the carotid artery, and so close to it that it is separated only by a thin layer of cartilage or fibrous tissue.

The tympanic orifice of the tube is as large in the child as in the adult. The Eustachian tube is more horizontal in the child, forming an angle of 10° with the horizon at birth, and an angle of 40° in the adult. At birth, the pharyngeal orifice of the tube lies below the level of the hard palate, in the adult it is about 10 mm. above it. It doubles its length between infancy and adult life, being 17 mm. in the former and 35 mm. in the latter ($\frac{5}{8}$ and $1\frac{3}{8}$ inches in length).

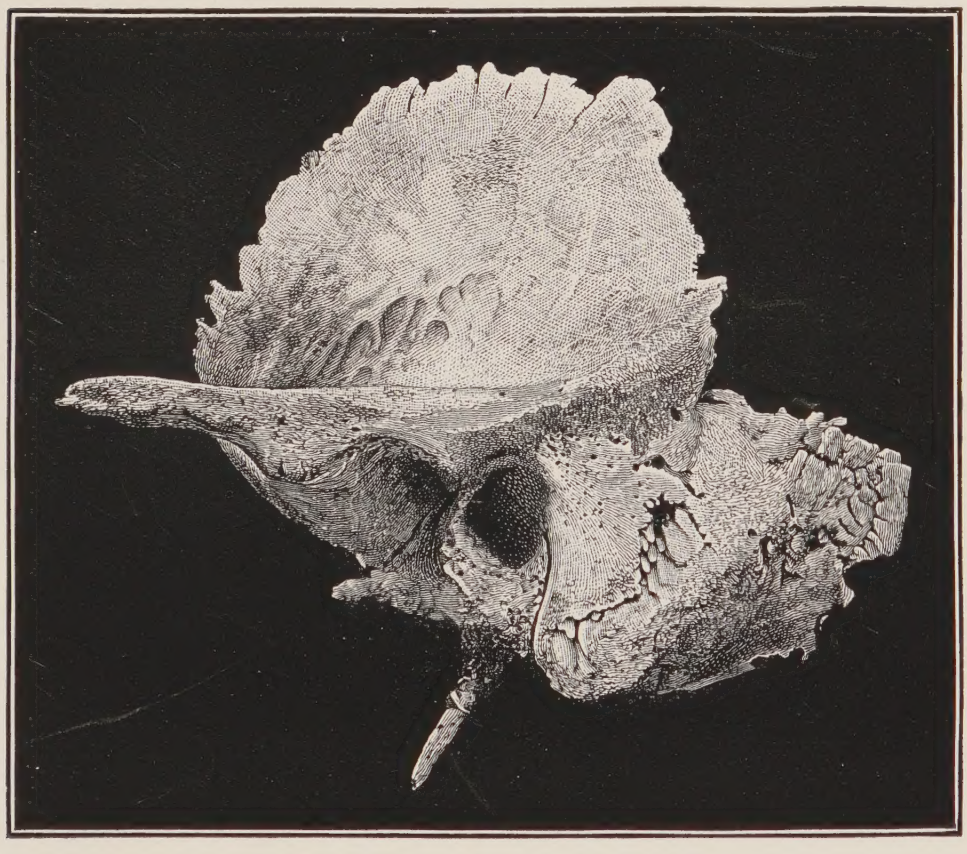
Besides being more horizontal in the child, the Eustachian tube is also shorter and wider, and consequently fluid escapes through it more readily from the tympanum during early life.

These facts are important in relation to the question of the escape of pus from the Eustachian tube, and also of the spread of disease from the pharynx into the tympanum. The ciliated epithelium, which lines the Eustachian tube and partly the tympanic cavity, has the movement of the cilia toward the pharynx. But pathological processes may soon lessen the activity of the cilia and inflammation may remove them altogether.

The *mastoid process* of the temporal is present at birth, and becomes pronounced externally about the second year. The mastoid antrum is found at birth, though the air cells in the mastoid are not developed till after puberty, their future position being, however, marked by cancellated tissue. Many of these cells open into the mastoid antrum and some directly into the tympanum. The upper layers of the mastoid cells have prolongations of the mucous membrane projecting into them from the antrum, and some from the tympanal cavity. The mastoid cells in after life vary greatly in size and communicate with each other.[2] Though the lower ones

[1] See Atlas of Head Sections, Plates 4, 5, 6, 14, 15, 30, 31, 38, 39, 46.
[2] See Atlas of Head Sections, Plates 18, 19, 20, 25, and 26.

FIG. 9.—LEFT TEMPORAL BONE, MALE 15 YEARS—MASTO-SQUAMOSAL SUTURE.



Left temporal bone of male, 15 years, showing masto-squamosal suture, dividing the mastoid rom the descending plate of the squamous.

To face page 12.

are not connected with the antrum by a mucous layer, yet, in the presence of molecular disintegration the intervening structures are destroyed, and the contents of the upper mastoid cells and the antrum penetrate into the lower ones, which become filled with granulation tissue and putrid pus. Only a thin osseous partition separates the sigmoid groove from the mastoid cells (as sections show).[1] Occasionally the osseous partition is defective, the wall of the sinus being then separated from the cells by a layer of fibrous membrane; it is even said that in some instances the mucous lining of the mastoid cells is brought into contact with the dural coat of the sinus. The osseous wall separating the mastoid cells from the groove of the sigmoid at its anterior knee is freely perforated by minute veins forming a communication between the sinus and the antrum. Occasionally the mastoid process is double. It remains quite separate in some animals, and it has been found separate on rare occasions in the adult human skull. It is said that emphysema may occur by escape of air from the mastoid cells into the subcutaneous tissue covering them, when a basal fracture has traversed them.

The *petrous bone* may be described as a prostrate three-sided pyramid, the base of which looks outwards and abuts upon the squamous and mastoid, while its apex is directed obliquely forward and inward.[2] A guide to the outer extremity of the posterior superior border of the petrous may be found on the exterior of the skull at the parieto-squamo-mastoid junction. A line drawn from this point to the external angular process of the frontal, on the opposite side of the cranium, gives the direction of the posterior superior border of the petrous, in line with the superior petrosal sinus. A rough indication of the direction of the border may be obtained by drawing a line from the upper and posterior margin of the mastoid process to the external angular process of the opposite frontal. The sharp bend which the carotid canal forms in the petrous assists to break the jerk of the column of blood passing through the artery. Fractures of the petrous involving the carotid canal have been known to cause rupture of the carotid artery, causing fatal extravasation.[3] Caries of this portion of the petrous has involved this canal, forming a distinct erosion

[1] See Atlas of Head Sections, Plates 8, 9, 34, 35, 40, 42, 44, 45, and 46.
[2] Leidy, *Science*, 1883. Vol. i., p. 507.
[3] Guthrie, *Injuries of the Head*, p. 73.

through its wall, and producing thrombosis of the carotid artery. The *fossa subarcuata* (floccular fossa *hiatus subarcuatus*).—On the posterior surface of the petrous bone, situated between the groove for the superior petrosal sinus and the internal auditory meatus, is an irregularly formed opening, which, though small and often obliterated in the adult, is yet a wide opening in childhood, penetrating the petrous under the superior semicircular canal, and ending in a series of cells under the last-mentioned structure[1] (Figs. 2 and 11).

In the fœtus of five months its orifice is quarter of an inch[2] in diameter, and the canal passes through the petrous portion, opening behind the auricle on the outer surface of the rudiments of the future mastoid process, and being separated from the skin by cartilage. At birth it still ends externally in this manner, but soon thereafter becomes closed within a bony mass, which renders it in the adult a narrow fissure ending in some cells under the superior semicircular canal, though it sometimes ends in the upper surface of the pyramid.[3] A vein passes through this canal surrounded by a fold of dura mater. In many of the mammalia the fossa sub-arcuata contains brain matter, and in them it persists through life. Both Gruber and Voltolini have demonstrated on the cadaver the extension of purulent inflammation by this channel from the mastoid cells to the cranial cavity.

The *facial canal* (aqueductus Fallopii), lying entirely within the petrous bone, has an irregular course which may be divided into three portions, according to its various directions.[4] First, it passes outwards toward the inner wall of the tympanum as far as the hiatus Fallopii. Secondly, at the position of the geniculate ganglion it turns at an acute angle, running in an oblique direction backwards, downwards, and outwards along the inner wall of the tympanum, appearing as a slight convexity at the posterior part of that cavity between the foramen ovale and the tympanic

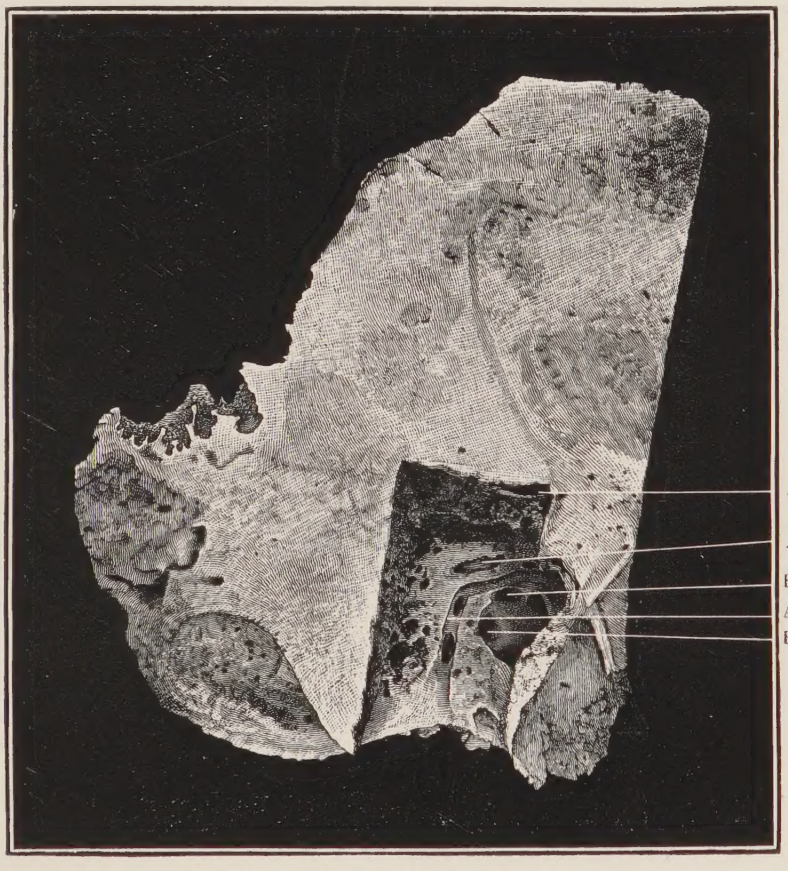
[1] In tracing this canal in two specimens of adult petrous bones, the author found it ending in both cases in cells in the petrous bone, underlying the superior semicircular canals. He also found that in one case it bifurcated toward its distal extremity. In a fœtal bone which he examined the canal was found to run very near to the external surface of the mastoid behind the ear. Voltolini describes this canal as ending even in the adult in the mastoid cells (canalis petro-mastoideus).

[2] (7 mm. × 5 mm.)

[3] Gruber, *Lehrbuch der Ohrenheilkunde*, Wien, 1888, p. 8. Voltolini, *Monatschrift für Ohrenheilkunde*, Jahr II. No. 2.

[4] See Atlas of Head Sections, Plates 16, 17, 23, 33, and 44.

FIG. 10.—PREPARATION OF RIGHT TEMPORAL BONE SHOWING THE COURSE AND RELATIONS OF FACIAL CANAL.



Mastoid portion of the right temporal adult bone, viewed from without. A block of bone has been removed showing the facial canal from the styloid foramen to its bend at the junction of the antrum with the tympanic cavity. In the shadow the fenestra ovalis and fenestra rotunda are seen. The external or horizontal semicircular canal presents itself above the posterior knee of the facial canal. A portion of the tegmen antri has been removed above so as to expose the middle fossa of the skull. The inner wall of the mastoid antrum has been partly removed, but the dark shading between the aperture into the middle fossa and the horizontal semicircular canal represents the portion remaining. The depth of the cavity produced by the removal of the block of bone measures at its upper part three-quarters of an inch from the surface to the deepest part of the aqueductus Fallopii.

To face page 14.

roof. The outer portion of the canal in the second part of its course may be seen in the passage between the antrum and the tympanum, about the middle of which passage it becomes superficial. Lastly, it then bends at an obtuse angle to form the third part, beginning in the floor of the antral passage near the posterior wall of the tympanum, at the level of the foramen ovale, and an eighth of an inch behind it, and descending with a fairly straight course to the stylo-mastoid foramen (Fig. 10). The facial canal in the new-born infant, as it passes through the tympanum, is a thin diaphanous plate, often having a defect of considerable size as it passes over the foramen ovale (Figs. 1 and 3). Hence in children facial paralysis is much more frequent, consequent upon disease of the tympanum extending through this hiatus, or through the very thin plate which divides the Fallopian canal from the tympanic cavity. In adult life an hiatus in the horizontal part of the Fallopian canal is frequently found, probably due to developmental arrest.

The mastoid antrum lies behind the facial canal, which descends in a line with the passage between the tympanum and antrum, but on its inner side. In order to avoid injuring the facial canal during operations, one must keep to the outer wall of the antral passage. The descending or mastoid portion of the facial canal lies at a variable distance from the outer surface of the mastoid. In a boy of twelve years the facial canal at the junction of the second and third portions, above the fenestra ovalis, lay fully three-quarters of an inch from the outer layer of the mastoid; while its lower extremity at the entrance of the styloid foramen was about half an inch from the surface. Therefore, in exposing the mastoid antrum, the higher and closer to the posterior zygomatic ridge the opening is made, the more surely will the Fallopian canal escape injury. The bend between the antero-posterior and the descending portions lies between the oval window and the lower semicircular canal, the latter being fully one-sixteenth of an inch to the outside of the former. When the tympanic cavity is exposed, the pyramid and the fenestra ovalis are guides to the position of the facial canal which lies behind the first and above the second of these; a line coinciding with the base of the pyramid may be taken as indicating the direction of the descending portion of the facial canal. The second part of its course may be indicated by the direction of the roof of the oval foramen. When the pyramid is destroyed,

the posterior margin of the foramen rotundum, though it lies deeper and more to the inner side, yet serves as a fair guide to the descending portion of the facial canal. The facial canal consists of harder and more compact tissue than that which surrounds it in the passage between the antrum and the middle ear, especially during childhood, adolescence, and early adult life. In addition to its cylindrical form and definite course, this fact facilitates its detection during operation. It may be occasionally seen almost isolated, the pathological processes having destroyed the surrounding cancellated tissue, leaving the dense canal like a tube crossing from the first to the second part of its course. Occasionally even in adult life it may be invaded by molecular disintegration, and its contents inflamed or destroyed.

The *sigmoid groove*[1] extends to the jugular foramen from a point on the inside of the skull corresponding to the asterion. It follows a tortuous and irregular course, which may be fairly indicated by the two following lines:—The first from the asterion to the superior margin of the external osseous meatus, of which line the posterior two thirds correspond to the upper part of the sigmoid groove, which is also the more superficial. The second line from the parieto-squamo-mastoid junction to the tip of the mastoid process corresponds in its upper two thirds to the vertical part of the sigmoid groove. The knee of the sigmoid (Fig. 11)—its most anterior convexity—is variable in its position, but is generally on a level with the upper part of the external osseous meatus. The sigmoid groove is situated at a variable distance from the external auditory meatus, the tympanum and the exterior of the skull. The distance between the external osseous meatus and the sigmoid groove varies from one or two to thirteen millimetres. When the sigmoid groove advances in an anterior direction, it also projects nearer the exterior of the mastoid. Occasionally on exposing a large mastoid antrum, which was prolonged backwards, the sigmoid groove was found external to and in front of a vertical line passing through the centre of the antrum, though the groove lay at a lower level and more superficial than the antrum. In different individuals the breadth of the sigmoid groove varies from three-quarters to a quarter of an inch; and it also varies in different parts of its course, the lower part of the vertical portion, including the knee, and the horizontal part being

[1] See Atlas of Head Sections, Plates 8, 9, 18, 25, 32, 34, 35, 38 to 40, 42, 44 to 46.

FIG. 10A.—COURSE OF SIGMOID SINUS RELATIVE TO EXTERIOR OF SKULL.

Skull with lines indicating course of sigmoid sinus with portion of lateral sinus; dotted lines indicating course of descending portion of facial nerve; over it a shaded part above external osseous meatus, indicating position of supra-meatal triangle.

To face page 16.

the widest. The right sigmoid groove is generally wider and deeper, projects more outward, and advances further forward than its fellow on the opposite side. Possibly this may account for the alleged greater frequency of intracranial sequelæ following otitis media on the right side than on the left. In infancy and early childhood the parts are so small and so closely associated with one another that in opening the mastoid antrum great care must be observed in order to prevent encroachment upon the sigmoid. The anterior portion of the sigmoid groove is perforated by numerous foramina for veins; these are especially abundant at its knee and toward the base of the skull at the junction of the petrous and mastoid portions. In infancy, the sigmoid forms a very slight groove, the sinus resting upon a much flatter surface than in the adult. The vein in the mastoid foramen, and occasionally those in the anterior and posterior condylar foramina communicate with the sigmoid sinus.

Körner's measurements (*op. cit.*) from cranial index are as follows :—

With a cranial index 1·55 to 1·50, the thickness of the osseous wall of the sulcus transversus, in its thinnest parts on the mastoid process, amounts on an average on the right side to 7·83 mm., at the most to 9 mm., and at the least to 7 mm. ; on the left the average is 10 mm., the maximum is 11 mm., and the minimum 9 mm. ; with cranial index 1·49 to 1·40, the average on the right side is 8·22 mm., maximum 15 mm., and minimum 4 mm. ; on the left the average is 9·72 mm., maximum 12 mm., minimum 7 mm. ; with cranial index 1·39 to 1·30, average on the right 6 mm., maximum 14 mm., minimum 3 mm. ; on the left, average 7·86 mm., maximum 15 mm., minimum 3·50 mm. ; with cranial index 1·29 to 1·20, right side, average 5·05 mm., maximum 7·50 mm., minimum 2 mm. ; left side 7·34 mm., maximum 11 mm., minimum 1·75 mm. ; with cranial index 1·19 to 1·07, average right side 4·22 mm., maximum 5·50 mm., minimum 2·30 mm. ; on left side, average 5·50 mm., maximum 8·50 mm., minimum 3·10 mm.

The lateral sinus.—The position of the lateral sinus is indicated by extending a line from the superior border of the mastoid apophysis to the inion, or where the skull is exposed from the asterion to the inion. It ought to be remembered that the sinus, instead of running a straight course, is convex upwards in most cases, while in some there is an upward concavity. The sinus in cross section is triangular, the base, however, being convex outwards.[1]

The relations of the temporo-sphenoidal lobe to the temporal bone.—The anterior surface of the petrous bone looks forwards, upwards, and outwards, the superior petrosal sinus occupying the highest

[1] See Atlas of Head Sections, Plates 10, 11, 12, 31, 32, 33, and 34.

ridge on its posterior border, the superior semicircular canal being occasionally a few millimetres above that level. Taking a horizontal head section at the level of the superior petrosal sinus, a considerable portion of the temporo-sphenoidal lobe will be found to lie in the deep hollow in front of it. The deepest part of the fossa is at the foramen spinosum. The depth is, however, very variable, being in some skulls much greater than in others.[1] Over the floor of the middle fossa, the dura is closely adherent to the tegmen. The pia mater, with its arteries, and the arachnoid meshes, though separated by a layer of fluid, yet lie close to the dura. In relation to the antrum and tympanum, the floor of the fossa media occasionally projects outwards beyond these cavities, and in two instances met with by the author, the floor of the middle fossa actually overlapped the upper portion of the outer wall of the antrum, so that, in order to reach the antrum, one had to perforate obliquely upwards and inwards.

The arterial supply of the temporo-sphenoidal lobe is important, as infective processes extending inwards from the base of the skull occasionally involve some of the arterial twigs and their perivascular sheaths. The superior convolution is supplied by the middle cerebral artery, the lower part by the posterior cerebral artery.

Relation of cerebellum to posterior fossa. — The line of the lateral sinus indicates the external boundary between the occipital lobe and the cerebellum, but toward the middle line the cerebellum ascends above this plane between the cerebral hemispheres reaching nearly to the level of the orbital roof,[2] and also projects downwards into the spinal canal considerably below the foramen magnum[3] so that it embraces not only the medulla but in many instances even the upper part of the cord. (For further relations see paragraphs Sigmoid Sinus, and Condylar Veins.)

Nerves passing through the temporal bone.—The facial nerve passes into the internal auditory meatus along with the acoustic.[4] The facial then enters the Fallopian canal, from which it emerges at the stylo-mastoid foramen. The branches of the seventh, within the petrous, are from the geniculate gang-

[1] See Atlas of Head Sections, Plates 39, 40, 41, 42, 44, 45.
[2] See Atlas of Head Sections, Plates 48 and 49.
[3] See Atlas of Head Sections, Plates 18, 36, 37.
[4] See Atlas of Head Sections, Plates 16, 23, 40.

FIG. 11.—LEFT TEMPORAL BONE—INNER ASPECT. THE ANTERIOR KNEE OF THE SIGMOID GROOVE.

The left temporal bone of male, 15 years, viewed from within. The sigmoid groove with its anterior knee is well-marked. The stylet lies in the course of an abnormal aperture for a large vein issuing from the tympanic cavity and entering the sigmoid groove. The fossa subarcuata is seen above the internal auditory meatus.

To face page 18.

lion—first, the large superficial petrosal passes out by the hiatus Fallopii, along the anterior aspect of the petrous beneath the Gasserian ganglion, through the foramen lacerum medium to the under surface of the base of the skull, where it unites with the deep petrosal to form the Vidian nerve, which joins the spheno-palatine ganglion: then several twigs are given off—one to Jacobson's nerve, which, as the small superficial petrosal, passes outside of the hiatus Fallopii, lying over the tensor tympani muscle, and emerging from the skull between the great wing of the sphenoid and the petrous, on its way to join the otic ganglion. As the facial descends behind the pyramid, it gives a nerve to the stapedius muscle, and near the lower end of the Fallopian canal the chorda tympani is given off.

The chorda tympani emerges by a foramen on the posterior wall of the tympanum,[1] and crosses the upper part of the inner wall of that cavity, entering posteriorly above the pyramid, passing between the handle of the malleus and the incus, and making its exit by a small foramen—the canal of Huguier—whence it proceeds to join the lingual nerve. The Fallopian canal is also pierced by a branch from the vagus (Arnold's nerve), and by the stylo-mastoid branch of the posterior auricular artery which supplies the posterior part of the tympanic cavity and the mastoid cells.

The chorda tympani, which is covered by a fold of tympanic mucous membrane, is often affected and disintegrated by destructive diseases invading the cavity. When the tympanum is thoroughly cleared out, along with its ossicles, the nerve, if not already destroyed, is almost necessarily removed. If so, the sense of taste in the anterior two-thirds of the tongue on the same side will be lost. The tympanic plexus also disappears in middle ear disease.

The tympanic nerve (Jacobson's) ascends from the petrous ganglia of the glosso-pharyngeal, through an aperture between the jugular fossa and the carotid foramen to the inner part of the floor of the tympanum, where it ascends over the promontory. It gives off several twigs which unite over the promontory with branches from other nerves, chiefly from the carotid plexus of the sympathetic, forming thus the tympanic plexus. The latter supplies branches to the mucous membrane of the Eustachian tube, the fenestræ ovalis and rotunda, and mastoid cells.

Although in facial paralysis the most prominent symptom

[1] See Atlas of Head Sections, Plates 33, 39.

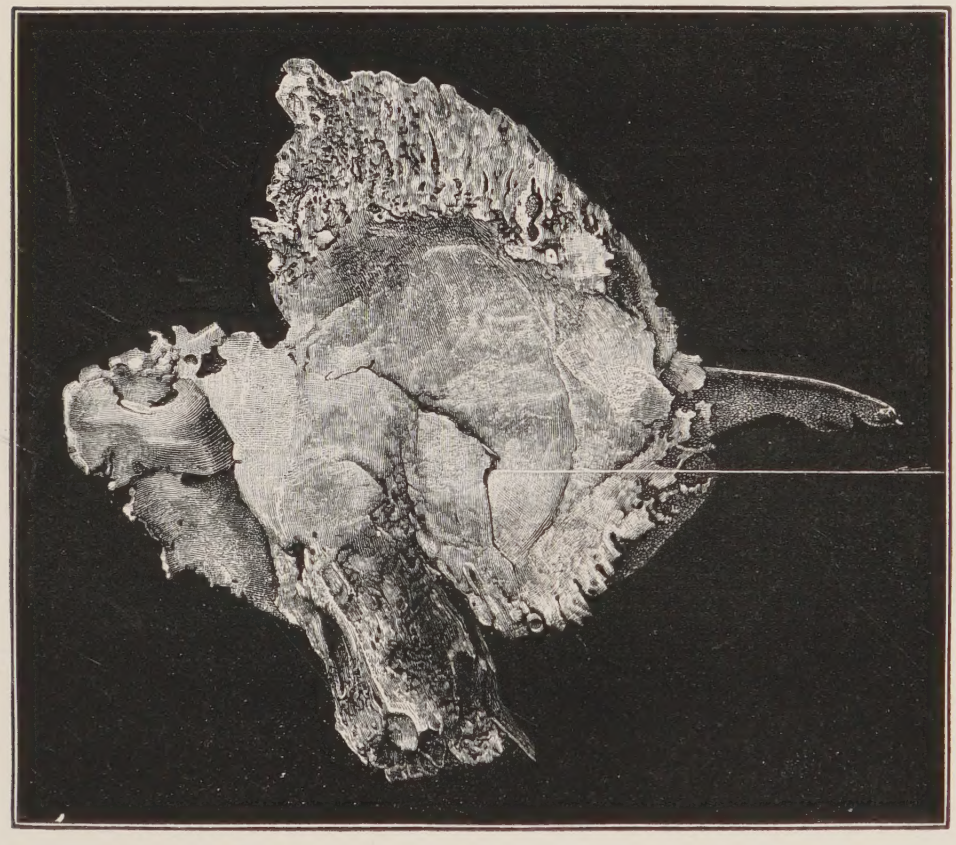
is the loss of motor power, causing a unilateral facial passivity —patients laughing and weeping as it were 'behind a mask'— yet its effects are more widely distributed. First, from exposure of the eyeball and from the want of proper distribution of the tears, conjunctivitis and epiphora are frequent. This conjunctivitis is passive as a rule, though when it occurs in weak children, who are neglected during the inflamed state of the eyeball, corneal ulceration is apt to occur, sometimes leading to permanent obscuration of the eyesight. Taste is affected through the chorda tympani. Smell is impaired from the want of power to dilate the nostril and from the dryness of the mucous membrane, owing to the imperfect conduction of the tears. Acoustic sensibility is increased (hyperakusis) from paralysis of the stapedius, permitting sound to act more vigorously on the stapes. Mastication is defective, and there is diminished secretion of saliva on the affected side.

Fibrillar contractions of the muscles of the face occur after destruction of the facial, and are to be regarded as a degenerative phenomenon, while the loss of secretion of sweat on the affected side is a sign of atrophy of the facial. As the facial is apt to be destroyed in young children the subjects of middle ear disease, it ought to be remembered that when this occurs in infancy during the active developmental period of the facial bones and muscles, arrest of growth of that side is liable to ensue, leading to further disfigurement. Section of the facial, in young animals, causes partial arrest of growth of the bones, muscles, and salivary glands.

In differentiating between paralysis of the facial muscles due to a nerve lesion and that due to a lesion in the facial cerebral cortex, the former is usually found much more complete. In the latter, the patient is generally able to close the eyelids voluntarily and retains a degree of emotional expression, while the sense of taste in the anterior two-thirds of the tongue is not affected.

Stimulation of the chorda in the tympanum causes prickling feelings in the tip of the tongue, while destruction of the nerve is followed by unilateral loss of taste in the anterior two-thirds of the tongue. Tactile and thermal sensibility are likewise said to be abolished over the same area, though this is not always apparent. It is supposed that the gustatory fibres of the chorda tympani originate, not in the facial, but in the glosso-pharyngeal, and may reach the chorda through the portio intermedia of Wris-

FIG. 12.—LEFT TEMPORAL BONE—INTERNAL ASPECT. SHOWING TEGMEN TYMPANI AND PETRO-SQUAMOSAL SUTURE.



The left temporal bone of a male, 15 years, viewed from within. Showing tegmen tympani with the petro-squamosal suture on its outer boundary. The mastoid foramen is seen at its junction with the sigmoid groove.

To face page 20.

berg. The secretions of the submaxillary and sublingual glands are also affected by division of the chorda, which supplies secretory and vaso-dilator fibres to these glands.

The *semicircular canals*, encapsuled in their hard bony covering, which has developed prior to and independent of the surrounding cancellated bone, are comparatively seldom involved in middle ear diseases, yet they are occasionally implicated, and may be encroached upon by operative procedures. Their section or injury does not interfere with hearing, but produces disturbances of equilibrium and sensations of giddiness. These are especially marked when the injury is bilateral. Pendulum-like movements occur in the direction of the plane of the injured canal. Thus, when the horizontal canal is injured, the head turns horizontally, especially when the person moves; when the posterior or superior are involved the pendulum movements are vertical, and the person has a tendency to fall backward or forward. When the whole of the canals are destroyed, standing may be impossible. It is, however, well to remember that intracranial section of the acoustic nerves gives the same result. Stimulation of the semicircular canals, causes nystagmus, the movements of the eyeballs following the plane of the special canal stimulated. The stimulation of one labyrinth affects both eyes. Violent injection of fluid into the ear of a rabbit causes vertigo, nystagmus, and rotation of the head towards the side injected, due to stimulation of the vestibular branch of the auditory nerve. Vertigo is often occasioned by injecting fluid into the human ear, from which the ossicles and the membrana tympani have been removed.

In one patient under author's care, there was great instability of equilibrium, evidenced by falling forward, while she experienced the sensation as if the earth "yawned" in front of her. She also had rotatory nystagmus. The semicircular canals were compressed by granulation tissue. After freeing the antrum and the cavity of the tympanum from granulation tissue, these sensations passed away, though the rotatory nystagmic movements were occasionally seen. When using injections into the middle ear, nystagmus occasionally occurs—much more frequent are vertigo and vomiting.

Perverted or defective ocular and auditory impressions may occasion vertigo. Giddiness and headache are often occasioned by paralysis of one of the ocular muscles, such as the external rectus, causing diplopia. Such vertigo is often accompanied by

vomiting. A hard plug of wax pressing on the membrana tympani may cause severe giddiness. These facts ought to be borne in mind in estimating the value of giddiness and vomiting in a supposed cerebral case.

The frontal sinuses[1] are situated on either side of the glabella, between the inner and outer tables of the skull, the bulging of their external wall forming the prominences of the superciliary arches. They are separated from each other by a mesial septum, often incomplete and occasionally wanting. Each frontal sinus communicates with the nose by a passage variable in size, which terminates in the anterior extremity of the middle nasal meatus. Their evolution occurs from the seventh to the twenty-first year of life, but they are occasionally developed at an earlier period. They are very variable in size and irregular in outline, in no two cases being entirely alike. In some skulls the frontal sinuses are scarcely discernible, while in others they form cavities capable of holding two or more ounces of pus. The sinuses are separated from the cranial fossa and from the orbital cavity by thin osseous lamellæ. They are lined with mucous membrane directly continuous through the infundibula with that of the nose, and are therefore prone to participate in the inflammations affecting the nasal mucous membrane. When inflammation attacks the frontal sinuses,[2] the lining membrane of the infundibulum becoming swollen occludes the intervening passage, and retention and accumulation of exudation ensues. The retained secretion escapes when the inflammation of the infundibulum subsides. The patient is, however, liable to recurrences of the empyema. From ulceration of the mucous membrane and osseous erosion, possibly in some cases aided by pressure, the walls give way at their weakest parts—the supra-orbital plates of the frontal bone—and the pus penetrates through the inner half of the upper eyelid. Hence such cases are generally seen in ophthalmic practice. The perforation follows this road in about ninety per cent. of the cases. The perforation very rarely occurs above the superciliary ridge. Erosion and perforation may, however, occur through the posterior wall of the frontal sinus, and pachy-meningitis be speedily established. In other cases the pyogenic organisms invade the pia mater or the brain, causing lepto-meningitis or cerebral abscess.

[1] See Atlas of Head Sections, Plates 1, 30, 41, 42, 45.
[2] See Cases LXI., LXII., LXIII.

The venous circulation.—The venous blood from the upper part of the cerebral hemispheres is collected by about a dozen vessels, the superior cerebral veins, lying in the sulci. These anastomose freely with one another, and with the inferior cerebral veins, and are joined by the veins from the mesial surface of the brain, just before they enter the superior longitudinal sinus. The external and inferior cerebral surfaces are drained by the inferior cerebral veins, which pour their blood into the lateral, the superior petrosal, and the cavernous sinuses. One of the largest of these veins, the middle cerebral, receives branches from the under surface of the frontal and temporo-sphenoidal lobes, and enters the cavernous sinus at the lower end of the fissure of Sylvius. The great anastomotic or Sylvian, another of these veins, runs forward in the posterior arm of the fissure of Sylvius, collecting blood from the surface of the parietal lobe and anastomoses with the upper cerebral veins. It perforates the dura mater near the lesser wing of the sphenoid, and passes backward in the middle fossa of the base of the skull, forming a communication with the middle meningeal veins, and pours its blood into the superior petrosal sinus. These are the two main channels by which the blood of the longitudinal sinus may communicate with the basal sinuses, the cavernous, and the superior petrosal. The middle meningeal veins, in the outer dural layer, from the area of distribution of the middle meningeal arteries, pass through the foramen spinosum to the pterygoid plexus. Several of the cerebral vessels have direct channels communicating with the middle meningeal veins. The corpus striatum and ventricles are drained by the choroid veins, which pass into the veins of Galen—the venæ magnæ Galeni—opening into the straight sinus.

There are two principal veins in the vicinity of the Rolandic convolutions; one, the great superior cerebral, running in the pre-Rolandic sulcus—occasionally in the Rolandic fissure itself—and communicating directly between the longitudinal sinus and the Sylvian vein. The great superior cerebral vein might be described as an ascending branch of the great anastomotic of Trolard. A second vein, of less size than the former, and running parallel to it in the post-Rolandic fissure, also unites the longitudinal sinus with the posterior aspect of the Sylvian vein, though occasionally it passes backwards to the lateral sinus. When it follows the usual course and joins the Sylvian, a vein passing backward and downward from the Sylvian joins the lateral sinus.

The *superior longitudinal sinus* receives its blood from the veins on the internal and external surfaces of the cerebral hemispheres.

The veins from the anterior lobe pass into the sinus in the direction of the stream, or, at most, at right angles to it; while the larger veins, issuing from the parietal and occipital portions of the brain, pass into the longitudinal sinus obliquely, from behind forward, so that their blood enters against the current in the sinus. The veins of the external surface of the cerebrum connect the superior longitudinal sinus above with the basal sinuses below, especially through the medium of the middle cerebral and great anastomotic. The veins of the frontal and parietal diploë pass into the longitudinal sinus, forming dilatations as they enter. Marasmic thrombosis of the longitudinal sinus is apt to originate in these dilatations. The middle meningeal veins communicate with those diploic dilatations, and consequently form connections between the longitudinal sinus and the veins of the pterygoid plexus, which empty themselves into the internal maxillary and deep facial veins. The longitudinal sinus also communicates with the scalp by the posterior parietal veins of Santorini. Organisms of erysipelas or other infective disease of the vertex may extend to the longitudinal sinus, through these veins. The longitudinal sinus is connected with the venous distribution of the mucous membrane covering the nasal septum, through the medium of a small vein penetrating the foramen cœcum. By this channel infective organisms, arising in lesions of the septum of the nose, may penetrate into the anterior part of the longitudinal sinus.

The sigmoid sinus—that portion of the lateral sinus contained in the sigmoid groove—receives blood from four sinuses, the longitudinal and the straight, through the lateral, the superior petrosal, and the occipital. It empties the main portion of its blood into the internal jugular, with which it is in direct continuity. The blood from both surfaces of the cerebellum and from the posterior part of the cerebrum is poured by venous channels into the lateral and sigmoid sinuses. Bloodvessels from the diploë of the skull enter it as they enter all other circumferential sinuses.

Numerous minute venous branches pass into the sigmoid sinus, while it receives groups of veins from the tympanum, the mastoid antrum and cells. These enter the sinus, especially at its anterior convexity or knee.

The lateral and sigmoid sinuses are usually asymmetrical, those of the right being generally the larger. The right sigmoid sinus is more marked than its neighbour, and its upper and anterior part has its convexity further forward and outward. These remarks apply especially to brachycephalic skulls.

It is supposed that the right lateral sinus generally receives the bulk of the blood from the superior longitudinal sinus, and the left lateral receives most of the blood from the straight sinus. The former would in that case receive the blood from the periphery of the brain and skull, while the latter would drain it from the central ganglionic portions of the brain.

The sigmoid sinus has direct communication with the exterior of the skull through two emissary veins, the mastoid and the posterior condylar. These veins are not constant and are subject to great variations, being frequently of considerable size.[1] When large on one side, they may be very small or absent in the other. The posterior condylar is the more constant and larger of the two.

The *mastoid vein*[2] passes from the sigmoid sinus in a backward and somewhat downward direction through the skull. Immediately to the outside of the cranium it joins an occipital scalp vein of considerable size, and the united veins pass into the deep cervical vein which joins the vertebral before it passes into the subclavian. Its course is, however, variable. It may divide into several branches as it passes through the skull, or extracranially it may join the posterior auricular vein, which passes into the external jugular. The first is the more constant course. When thrombosis occurs in the sigmoid sinus the mastoid vein is apt to become likewise affected. Even when the mastoid is thrombosed, if the thrombus does not extend beyond the thickness of the skull, it cannot be perceived during life, as the large scalp vein which it joins on the outside of the cranium continues patent, and of nearly normal dimensions. Erichsen mentions a case in which pus formed in the cerebellar fossa outside of the sigmoid sinus, and penetrated through the mastoid foramen, and formed an abscess on the exterior of the skull and the upper part of the neck. A mastoid vein distended with firm thrombus may fill

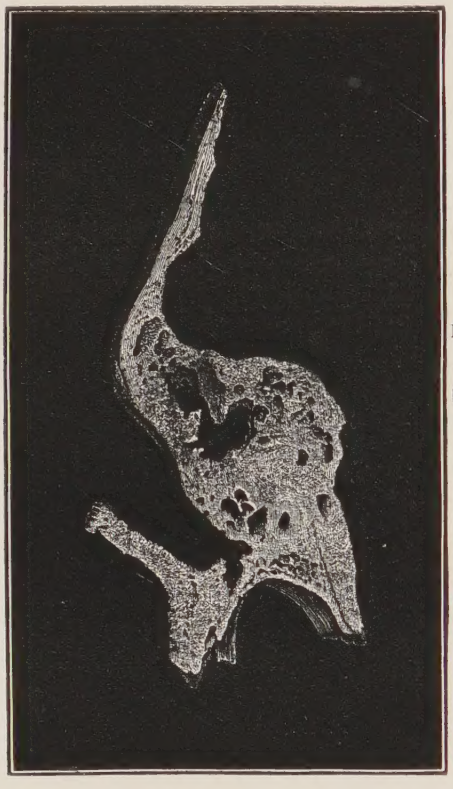
[1] See Table, pp. 27 and 28.
[2] See Atlas of Head Sections, Plates 34, 39.

its osseous canal so completely as to leave little room for pus to escape through it. Where pus does escape by the mastoid foramen, one can conclude that there exists extradural pus in the cerebellar fossa about the sigmoid groove, and, therefore, the probability is that the sinus is thrombosed. This latter condition will be rendered more probable by finding the mastoid vein thrombosed. There is in such a case the possibility of abscess in the cerebellum, though there may be only cerebellar extradural abscess, which may even have extended from the middle fossa of the skull, undermining the dura, separating the superior petrosal from the bone, and collecting round the sinus in the sigmoid groove. In one such instance under the author's care, an abscess existed in the base of the temporo-sphenoidal lobe, besides which there was suppurative pachy-meningitis, which had extended from the tegmen tympani to the cerebellar fossa, and by surrounding the sigmoid sinus had caused thrombosis. Suppuration to a slight extent was found in the inner half of the mastoid foramen, along with a thrombosed mastoid vein. There was no pus at the external orifice of the mastoid.

The *posterior condylar foramen* is occupied by a vein which connects the sigmoid sinus with the deep veins at the back of the neck, and the vertebral plexus.[1] Hence, when the posterior aspect of the petrous bone is covered with purulent secretion accompanying suppurative thrombosis of the sigmoid sinus, this foramen offers facilities for the drainage of the cerebellar fossa to the outside of the skull. When this occurs, the cellular tissue in the upper part of the posterior cervical triangle may become inflamed, and an extensive abscess may form under the deep cervical fascia secondarily to the intracranial suppuration. The writer has seen an instance of large abscess (situated in the upper third of the posterior cervical triangle exposing the occipital portion of the base of the skull) which had formed secondarily to an extensive cerebellar pachy-meningitis, accompanied with suppurative lepto-meningitis. Short of abscess, there is often a degree of inflammatory action in this region which produces tenderness on pressure, occasionally so acute as to cause the patient to cry out when touched. This is also a sequela of pachy-meningitis in the cerebellar fossa, and it frequently accompanies inflammatory thrombosis in the internal jugular vein.

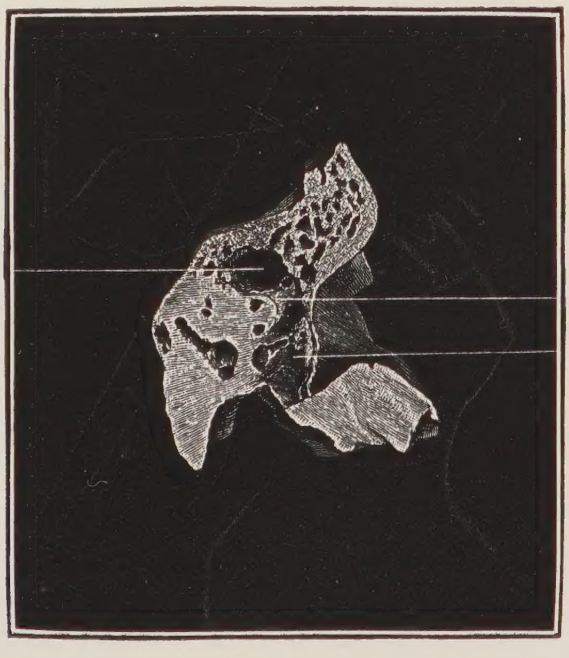
[1] See Atlas of Head Sections, Plates 31 and 36.

FIG. 13.—SECTION OF RIGHT TEMPORAL BONE SHOWING RELATIONS OF MASTOID ANTRUM.



Coronal section through the temporal bone, showing the mastoid antrum in its relations to the middle cranial fossa, the external auditory meatus, and a small portion of the middle ear toward its posterior aspect.

FIG. 14.—SECTION OF LEFT TEMPORAL BONE SHOWING COMMUNICATION BETWEEN MASTOID ANTRUM AND MIDDLE EAR.



Section of the petro-mastoid portion of the temporal bone viewed from before. Showing the posterior wall of the external auditory meatus, with the ridge for the attachment of the posterior segment of the membrana tympani, the passage uniting the middle ear and the mastoid antrum marked by the ridge of dense bone enclosing the Fallopian canal.

To face page 26.

Accompanying the hypoglossal nerve in the *anterior condylar foramen*[1] there are generally numerous minute veins from the membranes, and in cases of suppurative lepto-meningitis (consequently within the dura mater) this foramen may form an outlet for the dissemination of micro-organisms from within, or for the escape of pus. Occasionally this foramen is very large (see Fig. 21), and besides containing its intradural structures, it gives passage to a large extradural vein continuous with the sigmoid sinus. When this occurs, it frequently happens that the posterior condyloid foramen is absent or small, the vein which passes through a part of the anterior condylar foramen probably forming a substitute for the posterior condylar vein.

Fluid within the cerebellar dura mater readily finds an entrance into the spinal canal. Thus blood and pus from the cerebellar fossa may be found at the lowest part of the spine, having gravitated downwards.

The following measurements of the anterior and posterior condylar foramina were taken of four skulls in author's possession. These, along with the measurements given in the following table, show that in those examined *the foramina* were larger than is generally credited :—

I. Adult skull, in which right sigmoid sinus was comparatively narrow and apparently branched at the posterior condylar foramen :—

	Internal orifice.	External orifice.
Ant. condylar foramen,	8 × 5 mm.	8 × 4·75 mm.
Post. „ „	4·25 × 4 „	4 × 3·75 „

II. Temporal bone showing very deep sigmoid sinus and large mastoid vein. The antero-posterior depth of the sinus at level of junction of superior petrosal sinus with it = 15 mm. ($\frac{5}{8}$ inch).

Mastoid canal int. orifice—axis major 5 mm., minor 4 mm.

This canal divides in the substance of bone, into several diploic veins, one of which penetrates outer part of skull by a comparatively small external orifice for mastoid vein.

III. Bone with large ant. and post. condylar foramina :—

	Internal orifice.	External orifice.
Ant. condyloid,	7 × 4 mm.	6 × 4½ mm.
Post. condylar for.,	5 × 3·5 „	3·75 mm.

IV. Bone with large anterior, and no posterior condylar foramina :—

	Internal orifice.	External orifice.
Ant. condylar,	5 × 4 mm.	6 mm.

[1] See Atlas of Head Sections, Plates 8, 16, 23, 38.

Summary of measurements of condylar and mastoid foramina, in 225 skulls in the Glasgow Hunterian Museum and in the collections of Professors Cleland, Young and author, made by Dr. Thomson :—

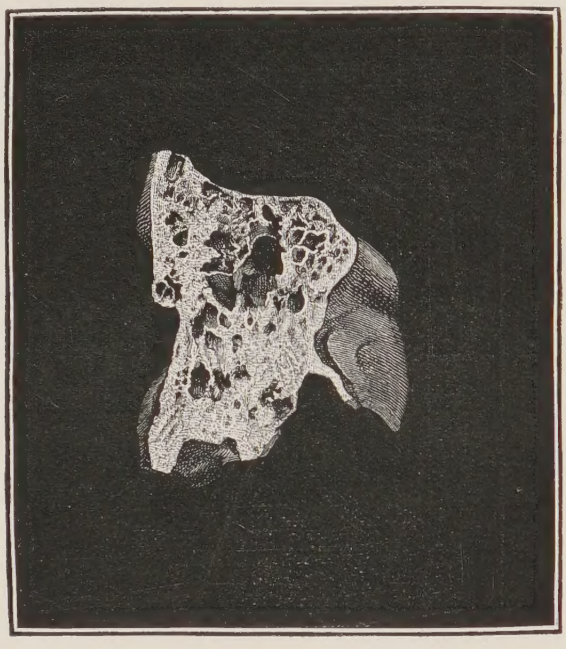
Diameter of *external* orifice of *right* anterior condylar foramen, - -	Average,	6·03 mm.
	Maximum,	11·00 ,,
	Minimum,	3·00 ,,
Diameter of *external* orifice of *left* anterior condylar foramen, - -	Average,	5·98 ,,
	Maximum,	12·00 ,,
	Minimum,	3·00 ,,
Diameter of *internal* orifice of *right* anterior condylar foramen, - -	Average,	5·86 ,,
	Maximum,	11·00 ,,
	Minimum,	3·00 ,,
Diameter of *internal* orifice of *left* anterior condylar foramen, - -	Average,	5·82 ,,
	Maximum,	11·50 ,,
	Minimum,	3·00 ,,
Diameter of *external* orifice of *right* posterior condylar foramen, - -	Average,	2·52 ,,
	Maximum,	8·00 ,,
	Minimum,	0·00 ,,
Diameter of *external* orifice of *left* posterior condylar foramen, - -	Average,	2·07 ,,
	Maximum,	9·00 ,,
	Minimum,	0·00 ,,
Diameter of *external* orifice of *right* mastoid foramen, - - - -	Average,	1·96 ,,
	Maximum,	5·50 ,,
	Minimum,	0·00 ,,
Diameter of *external* orifice of *left* mastoid foramen, - - - -	Average,	1·77 ,,
	Maximum,	4·50 ,,
	Minimum,	0·00 ,,

In 10·2 per cent. the *right* posterior condylar foramen was absent.
In 17·3 ,, ,, *left* ,, ,, ,,
In 4 ,, ,, *both* ,, ,, foramina were absent.
In 8 ,, ,, *right* mastoid foramen was absent.
In 7·5 ,, ,, *left* ,, ,, ,,
In 5·3 ,, ,, *both* ,, foramina were absent.

The position of the *sigmoid sinus* relatively to the *cerebellum* is important in connection with cerebellar abscess. The upper part of the sinus lies at the junction of the anterior and middle third of the cerebellar hemisphere, and lower, as it runs toward the apex of the petrous bone, it is further forward. Many of the head sections[1] show these relative positions as does a specimen in which the infective thrombosis of the sigmoid sinus stained the cerebellar hemisphere (see Fig. 52).

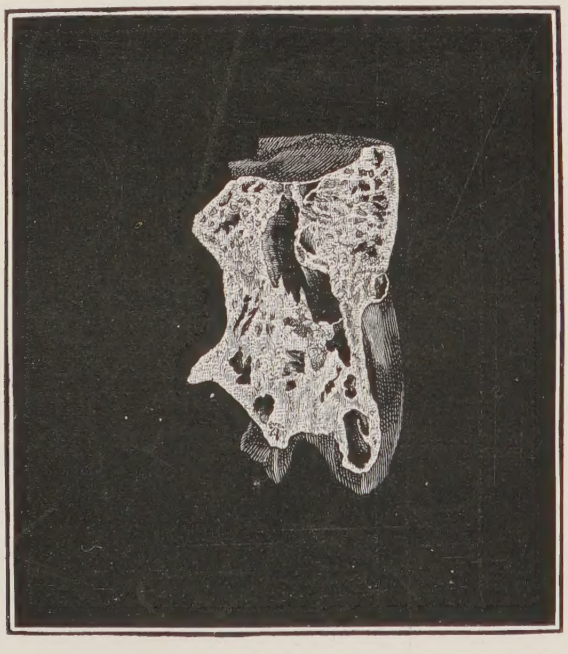
[1] See Atlas of Head Sections, Plates 8, 9, 18, 19, 26, 27, 31 to 34, 38 to 41, and 44 to 46.

FIG. 15.—SECTION OF LEFT TEMPORAL BONE SHOWING THE MASTOID ANTRUM AND CELLS.



A second coronal section of the petro-mastoid, viewed from behind, parallel with and quarter of an inch posterior to the section shown in Figure 14, representing the mastoid antrum in relation to the mastoid cells and middle cerebral fossa.

FIG. 16.—SECTION OF LEFT TEMPORAL BONE SHOWING MASTOID ANTRUM AND CELLS.



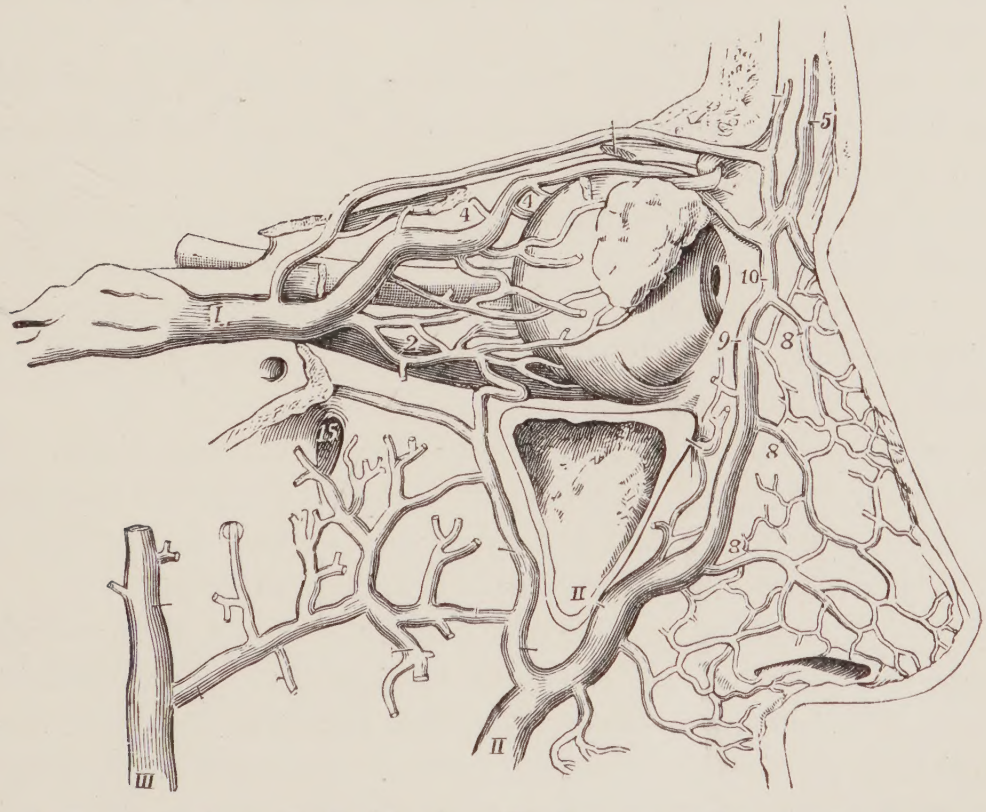
A third coronal section through the petro-mastoid, viewed from before, one-eighth of an inch posterior to section shown in Figure 15, three-eighths behind that represented in Figure 14, and one-eighth in front of the sigmoid sinus. Showing the posterior aspect of the mastoid antrum and a row of deep mastoid cells extending to tip of mastoid process with which the antrum is in direct communication.

To face page 28.

The cerebellum may be exposed on either side of the sigmoid sinus, preferably, however, posteriorly to it, as there is more room. The cerebellar abscess may have leaked through the dura mater, at the anterior and inner aspect of the sigmoid, in which case the opening would require to be made anteriorly to the sinus.

The cavernous sinus extends from the apex of the petrous portion of the temporal to the sphenoidal fissure, and through the circular sinus it anastomoses freely with its fellow on the opposite side. It receives the ophthalmic veins, which in turn anastomose with the facial through the nasal, and with the pterygoid plexus by the communicating ophthalmic, passing by way of the spheno-maxillary fissure. The inferior cerebral veins

FIG. 17.—ANASTOMOSIS OF CAVERNOUS SINUS WITH EXTRACRANIAL VEINS.



I. Cavernous sinus. II. Facial vein. III. Temporo-maxillary trunk. 8. Nasal veins. 9, 10. Angular veins. 2. Inferior ophthalmic veins. 4. Superior ophthalmic veins. 5. Frontal vein. 15. Spheno-palatine vein. (From Quain, after Hirschfield and Leveillé.)

empty themselves into the cavernous sinus, especially by the middle cerebral vein, and so does a small vessel—the spheno-parietal sinus—running inwards on the lesser wing of the sphenoid, which communicates with the middle meningeal vessels, and receives a branch from the dura mater. The cavernous sinus pours its blood chiefly into the petrosal sinuses, but has a communication with the pterygoid plexus through the Vesalian

vein which pierces the sphenoid bone. A small plexus from the cavernous sinus accompanies the carotid artery, opening into the internal jugular vein.

The inferior petrosal receives blood from the cavernous sinus, the inferior cerebellar veins, and those from the aqueduct of the cochlea, the vestibule, and the fossa subarcuata. It pours its blood into the jugular bulb, occupying the anterior compartment of the jugular fossa. It receives small veins through the foramen lacerum medium.

The superior petrosal extends from the cavernous sinus to the junction of the lateral with the sigmoid sinus. It receives blood from the inferior cerebral and superior cerebellar veins and small branches from the tympanum, many of which issue through the petro-squamosal suture, the remainder passing through the bone directly into the sinus.

The basilar plexus, over the basilar process of the occipital, opens into the inferior petrosal on both sides, and into the anterior spinal veins below.

The occipital sinus[1] communicates with the sigmoid and the posterior spinal veins. It receives several veins from the cerebellum and medulla oblongata. Its blood may find its way into the innominate, either through the vertebral vein, or through the jugular.

Most of the mastoid veins pass into the sigmoid sinus. The veins of the petrous bone pass partly into the sigmoid and partly into the petrosals. The tympanic veins pass partly into the middle meningeal, and in part into the sigmoid sinus.

There are four named diploic veins, the blood from which is poured partly intra- and partly extra-cranially. The frontal discharges the blood solely outside the cranium into the supra-orbital vein at the supra-orbital notch. The posterior temporal empties its blood entirely internally into the lateral sinus, while from the other two the blood is discharged in two ways: the anterior temporal externally into the deep temporal vein, and internally into the spheno-parietal sinus; the occipital externally into the occipital veins, and internally into the lateral sinus. Infective injuries to the diploë are apt to spread to the interior of the skull. Diploic diseases are prone to become disseminated in the skull. Tubercle, once it invades the diploë, spreads with rapidity, and suppuration in the diploë spreads readily into the

[1] See Schema of Intra- and Extra-Cranial Venous Anastomosis.

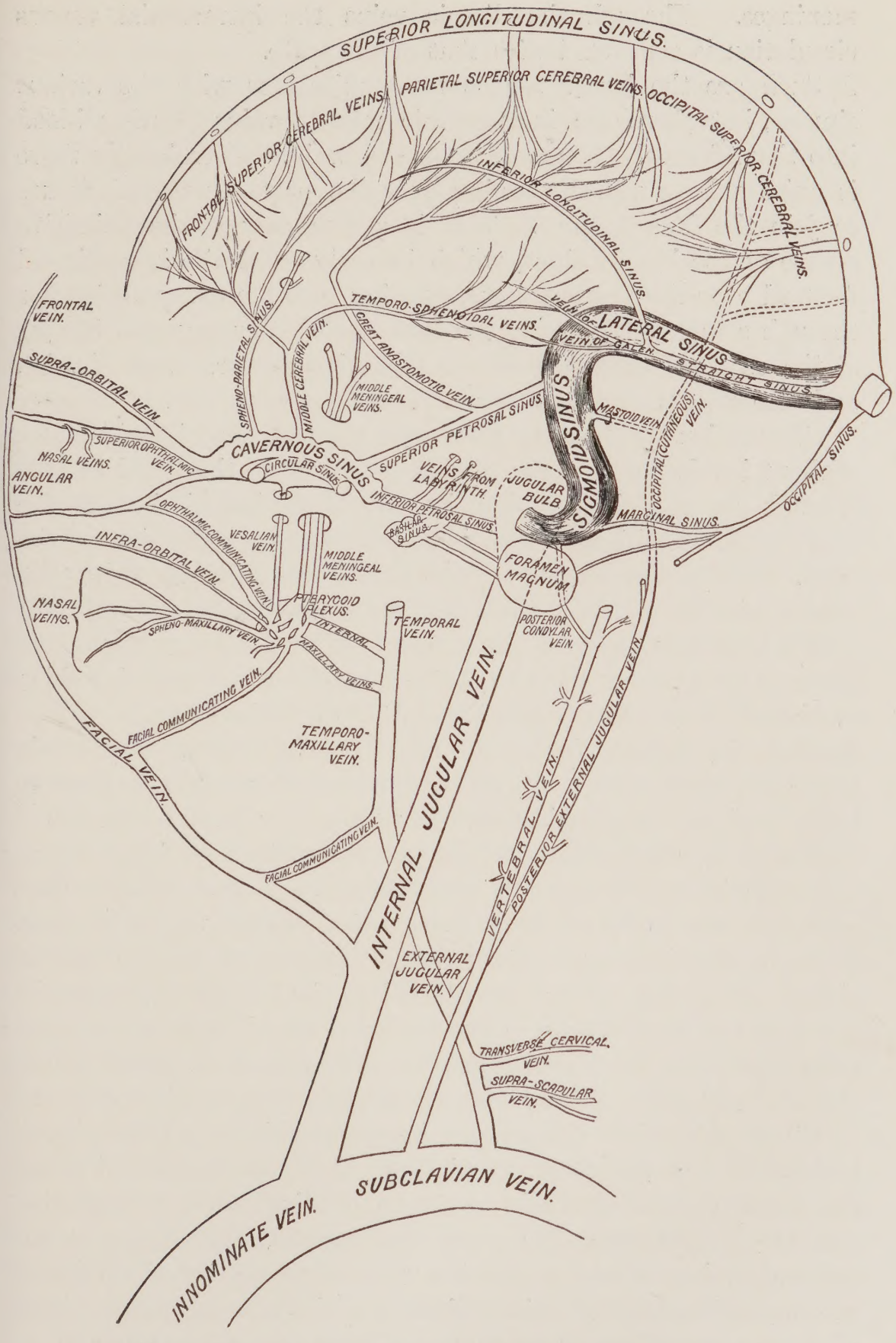


FIG. 18.—SCHEMA OF INTRA- AND EXTRA-CRANIAL VENOUS ANASTOMOSIS.

meninges. Through the diploic veins the intracranial venous circulation is connected with that of the scalp.

Such are the larger venous communications with the various sinuses, but there are innumerable minute veins pouring blood into them from the bone and from the brain. Occasionally there is quite a large vessel running over the petro-squamosal fissure to join the lateral sinus. This is a remnant of the primitive course of the lateral sinus, which in early fœtal life is continued forward over the petrous bone, opening into the external jugular through a foramen, piercing the base of the squamous, and issuing at the external extremity of the Glaserian fissure. See Fig. 12, and also Figs. 1 and 2.

Obstruction confined to the lower part of the sigmoid sinus, without involvement of the lateral, would cause the blood to flow through the mastoid vein, and by way of the lateral sinus to the Torcular Herophili, into the sinuses on the opposite side of the head. When the lateral is thrombosed continuously with the whole of the sigmoid, the blood in the superior petrosal will return into the cavernous sinus, from which the blood may cross through the circular sinus to the cavernous on the opposite side; or backward and downwards through the inferior petrosal; or forward by the ophthalmic; or downward through numerous channels to the pterygoid plexus. When the thrombosis occurs in the jugular bulb, leaving the sigmoid free, the return flow would occur to a slight extent, but the blood would pass through the occipital sinus, the posterior condylar vein, the vein through the mastoid foramen, and the diploic veins; the portion which ought to pass through the inferior petrosal would return to the cavernous sinus. Were the thrombus confined to the knee of the sigmoid the flow would still pass by the posterior condylar vein, the occipital sinus, and the internal jugular, the ligation of which would throw the blood in greater volume through these smaller vessels.

When extensive thrombosis has occurred in all the basal sinuses of one side, a reversed flow might be established from the lower part of the brain to the longitudinal sinus through the middle cerebral vein, the great anastomotic, and the superior cerebral veins, while a portion would escape by the middle meningeal, the spheno-parietal sinus, and through communications with the diploic emissary veins. Though the internal jugular is the main channel by which infective matter is conveyed from a thrombus of any part of the lateral or sigmoid sinus, yet it may

also be carried by way of the posterior condylar vein, the occipital sinus, and the vein through the mastoid foramen, all of which pour their contents indirectly into the subclavian and innominate, without passing through the internal jugular.

The *jugular foramen—foramen lacerum posticum*—is subdivided into three by fibrous processes, and in advanced adult life by bony spicules.[1] In the innermost compartment there are the inferior petrosal sinus and the meningeal branch of the ascending pharyngeal artery; in the middle are the glosso-pharyngeal, the vagus, and the two divisions of the spinal accessory nerve;[2] while in the most external there are the sigmoid sinus, and the posterior meningeal artery, which is a branch of the occipital. The petro-occipital ligament separates the external and most posterior compartment from the middle nervous one. When this ligament becomes ossified, it is named the intra-jugular process.

The cavernous sinus has on its inner wall the internal carotid surrounded by the carotid plexus of the sympathetic with the sixth nerve on the outer side of the artery; while on the outer wall of the sinus there are the third, fourth, and ophthalmic division of the fifth nerves.[3]

The pterygoid plexus (Figs. 17 and 18) is situated round the external pterygoid muscle, chiefly between its two heads, which converge to be inserted into the pterygoid fossa beneath the condyle of the lower jaw.[4] The intracranial blood is poured into the pterygoid plexus by the middle meningeal veins through the foramen spinosum, by the Vesalian piercing the sphenoid and communicating directly with the cavernous sinus, and by the communicating ophthalmic vein which escapes through the spheno-maxillary fissure. Besides receiving many muscular branches, it has an intimate relation with the venous circulation of the nose, three veins from the nasal fossa pouring their blood into it through the spheno-maxillary vein. The blood from the pterygoid plexus is conveyed through the facial vein and the internal maxillary veins into the jugulars (external and internal).

Mechanism preventing the aspiration of the sinuses.—The return flow of blood from the brain is poured intracranially into the sinuses, and these in turn empty their contents mainly into the jugular veins, the sinuses and those vessels being in direct con-

[1] See Atlas of Head Sections, Plates 16, 17, 36, 37, 44.
[2] See Atlas of Head Sections, Plates 16, 17.
[3] See Atlas of Head Sections, Plate 15.
[4] See Atlas of Head Sections, Plates 37, 38.

tinuity the one with the other. Though they are directly continuous, there is a marked difference between them. The sinuses are rigid non-collapsible tubes which ever remain patent, the veins are thin, delicate, flexible, curtain-like vessels, whose calibre during life is constantly varying from a state of almost complete collapse to one of great distension. If the carotid sheath be opened from its outer aspect so as to expose the internal jugular vein, one sees those fluctuations very markedly. One moment the vein presents itself as a turgid vessel, whose lumen is half an inch to an inch in diameter; the next instant it subsides, so that it appears as if its walls met as it becomes flattened like a ribbon against the underlying parts. The vein is turgid on expiration, and becomes almost collapsed on inspiration. Though this is best seen in the internal jugulars, the other cervical veins participate to an extent in this action, as may be seen in the external jugulars, especially when the patient is under chloroform. When this aspiration occurs in those large veins on the outside of the skull which adapt themselves so absolutely to the varying volume of their contents, what becomes of those intracranial rigid tubes—in direct continuity with the veins—whose lumen cannot vary? As their walls cannot yield, they are liable to be emptied on each inspiration. If they be regarded as parts of an ordinary hydrostatic mechanism, a series of tubes connected with a limited and slowly replenished reservoir—the vessels of the brain—then on each inspiration there would follow an aspiration, not only of the contents of the rigid tubes, but of that of the vascular system of the brain besides. Though dealing with an enclosed system of vessels yet they are of varying size, and the calibre of the terminal capillaries is far too limited to permit of them supplying on a sudden a volume of blood sufficient to prevent the emptying of the sinuses on sudden aspiration. Were the sinuses thus aspirated, the whole of the blood in the brain would be propelled suddenly forward to compensate for the volume withdrawn, and, consequently, there would be on each deep inspiration, a sudden faintness or want of brain power, which, on very deep inspiration, might produce something akin to momentary unconsciousness, and, at least, would cause distinct disturbance of brain function. The sudden emptying of the venous channels situated over the brain surface would produce on the brain substance a physical impression akin to shock. That this does not occur

shows that there must be some mechanism so arranged as to prevent the aspiration of those sinuses.

The whole intracranial venous arrangement is calculated to ensure an even, regular, constant flow of blood without fluctuation, and it is beautifully guarded from suction interference on the part of the large extracranial veins.

First—The blood in the longitudinal sinus is dammed back by the manner in which the larger middle and posterior cerebral veins enter the sinus, and pour their blood obliquely into it against the stream.

Second—The division of the sinus at the Torcular Herophili is such that the stream divides into two, which diverge at right angles to the original stream, and by this deflection the force of the current is broken.

Third—In the lateral sinus the stream follows a horizontal course with a convexity outwards as well as upwards, though sometimes it is downwards.

Fourth—In the first part of the sigmoid sinus the stream takes a quick bend to a vertical direction.

Fifth—It pursues a horizontal course from without inwards along the floor of the skull, with a marked convexity downwards.

Sixth—It then makes a quick turn, almost doubling on itself upward and outward, with a corresponding narrowing of its calibre just before it enters into the wide dome-shaped jugular fossa.

Seventh—This fossa is not only expanded above, but its osseous dimensions are narrowed toward its exit.

Eighth—Besides its osseous exit being narrowed, there is a further restriction of the space by the presence of the three nerves—9th, 10th, and 11th—and of the inferior petrosal sinus, all of which enter the jugular fossa. There is also, just at the exit, a small pad of cellular tissue, surrounding some minute veins, and covering the posterior aspect of the outlet of the fossa, which further narrows the space. This pad is somewhat elastic, and is capable of both expansion and compression, according to the varying states of the internal jugular vein.[1]

Ninth—The direction of the outlet of the jugular fossa is obliquely forward, while the course of the jugular vein beyond that is obliquely downwards. The vein will, therefore, impinge when emptying against the rim of the exit.

[1] See Atlas of Head Sections, Plates 36, 37, 38.

Tenth—The very tenuity of the walls of the internal jugular favours and assists in shutting off its connection with the sinuses. A thin elastic tube filled with water suddenly aspirated occasionally closes at one point from the bringing together of its walls, and immediately after the suction is removed its walls expand and the flow proceeds.

Eleventh—The digastric and the styloid muscles are in close proximity to the outer wall of the internal jugular vein, and it is possible that some of these muscles, especially the stylohyoid, may exert a slight pressure upon the jugular during inspiration.

The relative position of the last curve in the horizontal limb of the sigmoid sinus to that of the jugular bulb is of great importance (see Fig. 19), as the level of the fluid in the former is much lower than in the latter; so much so that the floor of the exit—as it

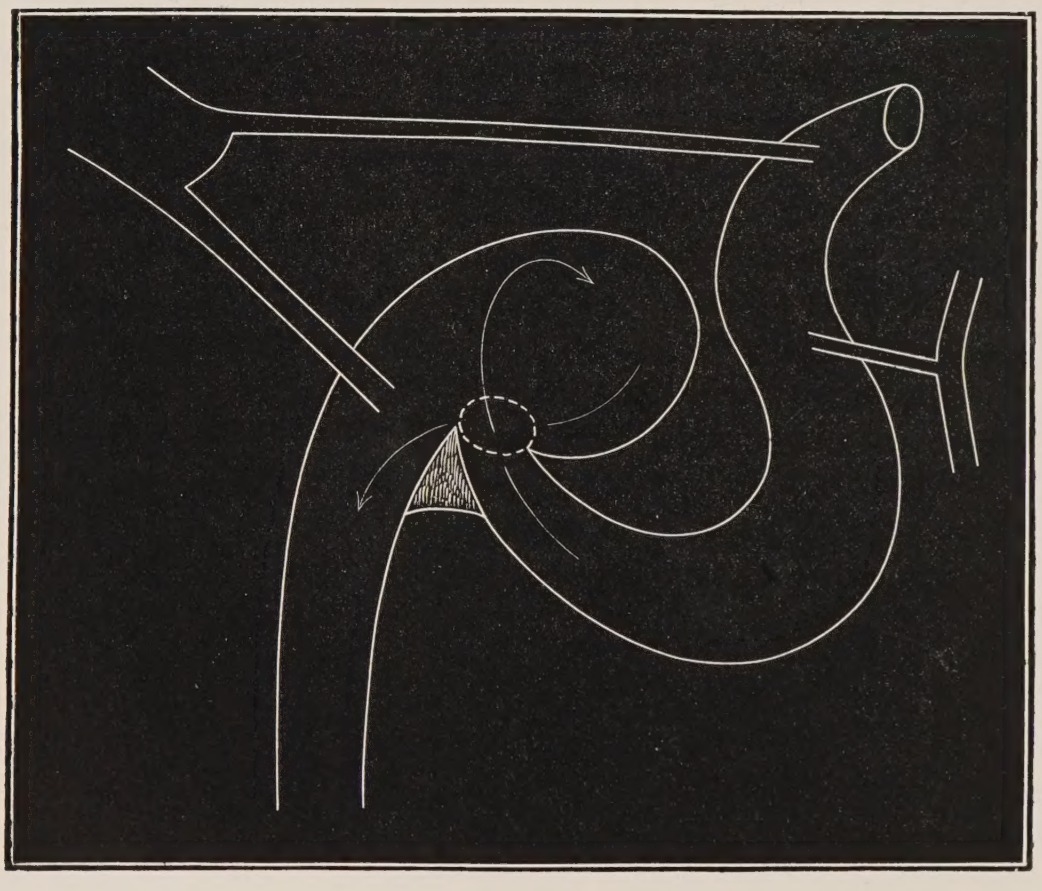
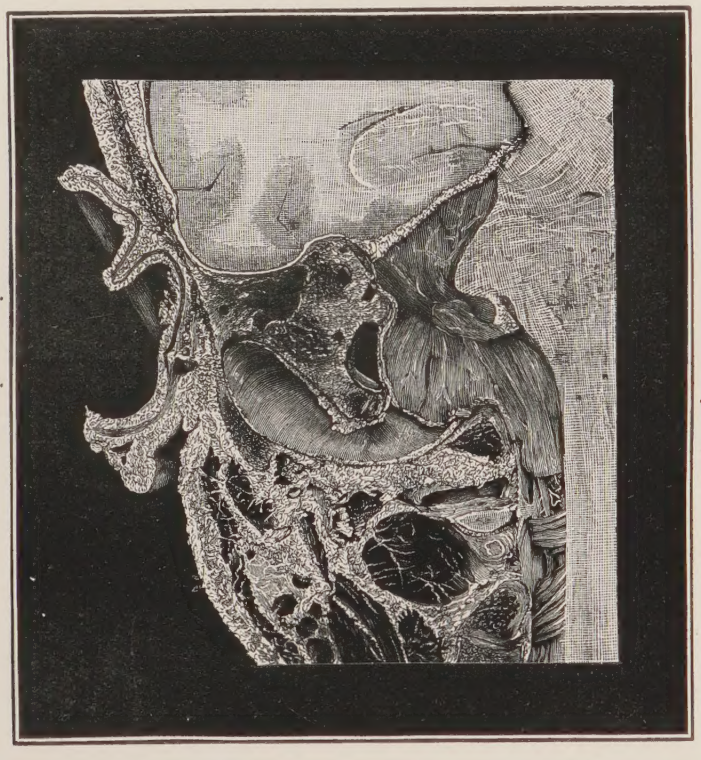


FIG. 19.—SCHEMA OF SIGMOID SINUS IN RELATION TO JUGULAR BULB.

passes into the jugular bulb—is on a level with the roof of the horizontal part of the sigmoid. Again, the roof of the jugular fossa is very much above that of the whole horizontal limb of the sigmoid

FIG. 20.—CORONAL HEAD SECTION—SIGMOID SINUS AND JUGULAR DOME.



Woodcut from photograph of head section, showing relative levels of jugular dome, vertical and horizontal portions of sigmoid sinus. The relations of the mastoid antrum to the knee of the sigmoid sinus are also illustrated.

To face page 36.

sinus (see Figs. 20 and 21, and also [1])—occasionally half an inch higher. Consequently, in the upright position, this limb of the sinus will contain a supply of fluid on a lower level than its exit, and therefore it will act as a retarding medium to the emptying of the vessel. The jugular bulb being always full and so much above the level of the outlet of the sinus, tends to dam back the flow from the sinus. The great width of the sigmoid sinus, compared with the lateral sinus, or the outlet of the sigmoid at its junction with the jugular, is such as to dissipate the force of the blood stream in the same way as the flow of a river lessens in force when it is suddenly expanded into a lake, *e.g.*, the Rhine and lake Geneva.

There is generally a mastoid vein which passes from the vertical limb of the sinus outward and backward through the skull, and when this vein is absent, other emissary veins act in the same way, such as that from the Torcular Herophili. On the outside of the skull the mastoid vein joins somewhat obliquely a large vein from the scalp which passes down the neck. This extracranial vein—one of the occipital or post auricular—with its continuous stream of blood from the scalp, flowing over the mouth of the mastoid vein, exerts a suction upon the latter, which will be communicated to the sinus and further retard its flow. Even if this external neck vein should participate in the collapse attending the internal jugular, which is improbable as it is too far removed from the direct aspiratory force produced by inspiration, it would still act upon the blood in the sinus as a deterring force—as if one placed a finger upon a pipette. Again, if this collapse of the internal jugular were absolute, which is very seldom, there might be a difficulty in re-establishing the flow. Here, again, there is a mechanism, which would prevent that possibility. The inferior petrosal sinus enters the jugular fossa and is constantly pouring its blood into that pool, so that the supply and the continuity of the stream with the sinus and jugular vein, are maintained. Not only so, but the inferior petrosal being a straight sinus, and having a sharp descent from a large venous reservoir—the cavernous sinus—the force with which its contents flow will in all probability be greater than that of the sigmoid sinus, when the latter pursues such a tortuous course, having the force of its current dissipated by its wide expansion, and having besides an ascent and narrowing of its lumen toward its exit in the jugular fossa. The

[1] See Atlas of Head Sections, Plate 44.

extra speed of the blood from the inferior petrosal sinus ensures the continuity of the flow from the sigmoid sinus into the jugular vein. It is also possible that the flow of blood through the inferior petrosals may be further hastened by the pulsations of the two internal carotids, acting on either side of the cavernous sinus like a hand pressing gently on an india-rubbber ball.

The various positions which the head may assume affect the mechanism, but they are met by the multiplicity of curves which serve in their turn the above function, and the principle of the mechanism is preserved.[1]

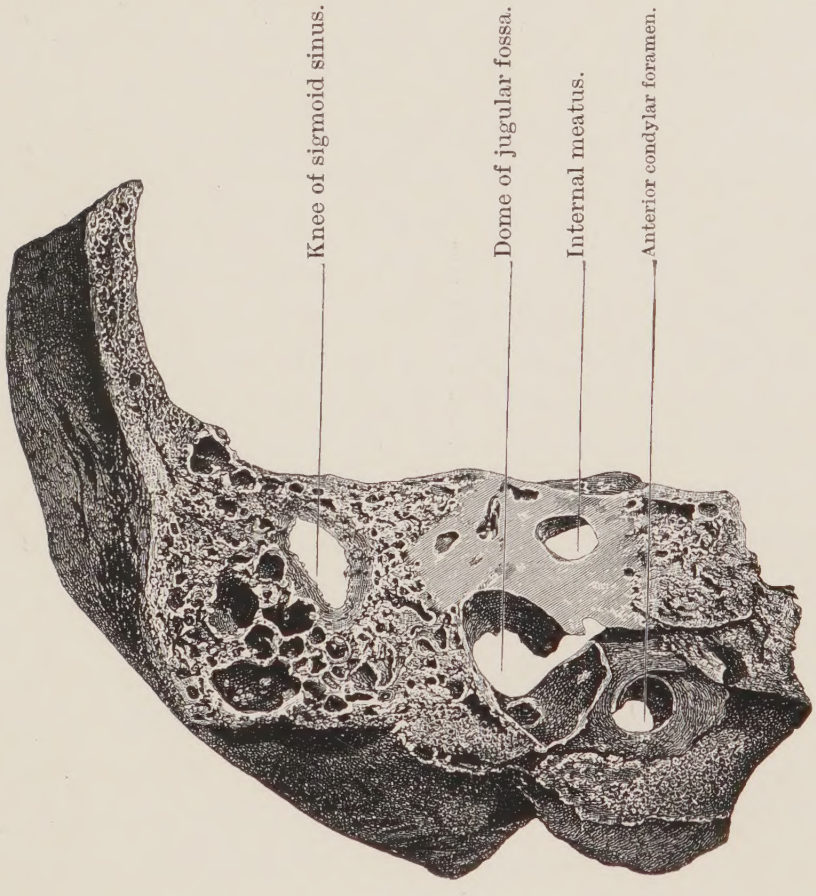
It is also interesting to note that the fœtal distribution of the vessels is different from that just described. At an early period of fœtal life the lateral sinus pours its blood into the petro-squamosal sinus, which in turn empties itself into the external jugular vein. In the fœtus there is no lung breathing, and consequently no aspiration. Therefore the special mechanism which would guard against aspiration is not required, and the passage of blood into the heart takes a less tortuous and more direct course.

Here, therefore, is a beautiful mechanism for retarding the flow of blood, and shutting it off from the aspiratory influence to which it would otherwise be subjected, so that the brain is guarded against the sudden loss of functional activity and faintness, which would otherwise be brought about by the sudden withdrawal from, and the constant variation of, the quantity of blood within it. At the same time, a strong aspiratory force is applied, which quickly withdraws the blood discharged from the sinuses, once it has escaped from the jugular fossa, and so prevents any impediment to the flow.

Lymphatics.—The lymphatics from the *scalp* pass into the occipital, the mastoid, and the parotid lymphatic glands, while those from the forehead join the facial lymphatic vessels. The lymphatics of the *face* are divided into two sets, a superficial and a deep. The former, following the course of the facial vein, terminate in the submaxillary and parotid lymphatic glands; the latter, including those of the orbit and nasal cavity, end in the internal maxillary glands. The *intracranial* lymphatics have their origin in the cerebral pia mater and in the choroid plexuses

[1] Mr. Blyth of Anderson's College, Glasgow, suggested the need of increased speed in the flow of the inferior petrosal in order to make the mechanism hydraulically perfect. Dr. Thomson thought the pulsations of the carotids might facilitate the flow through the cavernous sinus into the inferior petrosals.

FIG. 21.—SECTION THROUGH THE RIGHT PETRO-MASTOID AND PORTION OF THE OCCIPITAL BONE, SHOWING HEIGHT OF INTERNAL JUGULAR DOME IN RELATION TO THE VERTICAL LIMB OF SIGMOID SINUS.



Coronal section through petro-mastoid, showing mastoid cells, anterior portion of knee of sigmoid sinus, dome of jugular fossa, with posterior condylar foramen opening on its floor, the anterior condylar foramen of large size, and the internal auditory meatus.

To face page 38.

left in the permanent facial paralysis and the unsightly indurated of the ventricles. They pass out of the cranial cavity along the internal carotid and vertebral arteries and the internal jugular vein to the deep cervical glands. Others pass from the choroid plexuses of the lateral and third ventricles, coalescing into a lymphatic vessel which accompanies the veins of Galen. The superficial cervical glands lie in the course of the external jugular vein, between the platysma myoides and the deep fascia. Into these pass all the lymphatics from the integuments of the neck, the external ear, and the efferent vessels from the sub-occipital, mastoid, parotid, and submaxillary lymphatic glands, while the efferent vessels from the superficial cervical enter the deep cervical glands. Extracranial lesions of an irritative kind will first show themselves in the enlargement of one of the groups of sub-occipital, mastoid, submaxillary or parotid lymphatic glands; but, if the irritation be continued, the superficial cervical will enlarge, and if it still persists, and is sufficiently severe, the deep cervical will also participate in the enlargement. Intracranial irritation shows itself first in the swelling of the deep cervical glands, but as they lie under the cervical fascia, their enlargement is apt to be concealed unless specially looked for. By the involvement of the cervical glands, a meningeal lesion can be differentiated neither from a cerebral one nor from a thrombosis of the sigmoid sinus. The white swelling so frequently accompanying phlebitis and infective thrombosis of the sigmoid and the jugular is largely glandular. In one instance of acute localized tubercular affection of the mastoid and left side of the middle fossa of the skull, the posterior auricular and mastoid glands were implicated along with the deep cervical under the sterno-mastoid muscle in the sheath of the internal jugular.

In several instances of suppurative meningitis of the posterior fossa, the glands in the posterior triangle (sub-occipital) were involved. In three cases of *cario-necrosis* of the mastoid and contiguous petrous portion of the temporal, accompanied by profuse suppuration, a mass of enlarged glands were detected under the superficial layer of the deep fascia, behind the sterno-mastoid and partly covered by that muscle; and, in one instance, a large gland mass was dissected from the same situation, encroaching closely upon the sheath enclosing the internal jugular. Years after the primary lesion had subsided its traces were still

gland, which was removed as above mentioned. The relations of the cervical glands to the internal jugular vein are well shown in some of the plates of Sagittal head sections.[1] The value of glandular cervical enlargements in differentiating intra- from extra-cranial inflammation is dealt with under the chapter on "Symptoms."

Cerebral membranes.—There are three membranes usually described as covering the cerebro-spinal centres; an external fibrous one, the dura mater, which lines the interior of the skull and forms a loose sheath in the spinal canal; an internal, the pia mater, consisting of areolar vascular tissue, which closely invests the brain and spinal cord; and an intermediate non-vascular membrane, the arachnoid, which lies over the pia mater, in some places so closely connected with it as to be indistinguishable from it, in others separated from it by a considerable space.

The *dura mater cerebralis* is a strong dense tunic of considerable thickness, consisting of white fibrous and elastic tissue arranged in bands and laminæ. A layer of flattened endothelial (epithelial) cells covers its inner surface, and also in part its external surface, at these places where it is not adherent to the bones and the sutures. It is traversed by bloodvessels, which are chiefly found in its outer layers, and serve mainly for the nutrition of the bone. It is supplied by numerous nerve filaments from the fourth, fifth, and twelfth cranial nerves, and from the sympathetic. Its lymphatics are abundant. There is besides a wide meshed capillary network with peculiar dilatations near the inner surface of the cranial dura mater. The spaces between the fibrous trabeculæ contain flattened connective tissue corpuscles. These spaces serve for the passage of lymph, and they can be injected from the epidural space, where this exists, as in the spinal canal; and in the cranial dura the injecting fluid can be forced along them, through the thickness of the dura mater, into the subdural space. They can also be filled by inserting the injecting cannula into the substance of the membrane. This shows that the subdural space communicates with the lymphatics in the dura, while the latter communicate with the dural veins. Hence, once pathogenic organisms penetrate into the wide meshed capillary network on the inner side of the dura mater the whole of the subdural space is open to invasion. The cerebral dura mater adheres to the inner surface of the bones, forming

[1] See Atlas of Head Sections, Plates 31 to 35.

their internal periosteum, but its connection with them is of varied degrees of intimacy, depending greatly on the bloodvessels, and the fibrous processes which pass from the membrane to the bones. Over the vertex the dura is comparatively loosely attached except along the lines of the sutures, where it adheres more closely. A probe passed through a trephine opening on the vertex may be introduced between the skull and the dura mater for a considerable distance provided the sutures are avoided, and considerable extravasations of blood or pus may take place over the vertex, between the skull and the dura mater, though these are generally limited to one bone. The whole parietal dura may be thus stripped, the limits being the sutures. Though this is the rule the sutures do not always limit purulent collections; Case No. XXXVII. is an instance, as pus from the cerebellum had separated the tentorium and was found to occupy a large cavity between the skull and dura over the parietal and occipital bones, and communicated with an intradural abscess in the posterior segment of the temporo-sphenoidal and adjacent portions of occipital lobe.

At the base, which is irregular and perforated with numerous foramina, the dura mater is either prolonged to the outer surface, where it becomes continuous with the pericranium, or its fibrous tissue becomes blended with the areolar sheath of the nerves. Here the membrane is so closely adherent to the bones that it would be difficult for blood or pus to collect in any quantity between the basal dura mater and the skull. In consequence of the intimate relations between the base of skull and dura mater, fractures of the former are often accompanied by rupture of the latter, and occasionally cerebro-spinal fluid may escape through these apertures. Fractures of the calvarium when fissured are seldom accompanied by rupture of the dural membrane.

The fibrous tissue of the dura mater is divided into two distinct layers, which at certain parts separate from each other, leaving channels constituting sinuses, which are lined with endothelium, continuous with the internal coats of the veins. It is through these that the venous blood from the brain issues. They are unyielding, and admit of little distension or collapse, hence they exert an equal pressure, neither pressing unduly on the brain nor being pressed on by it. As they have little or no power of contracting or retracting, it is evident that they present a serious lesion when wounded, and hæmorrhage from them cannot be

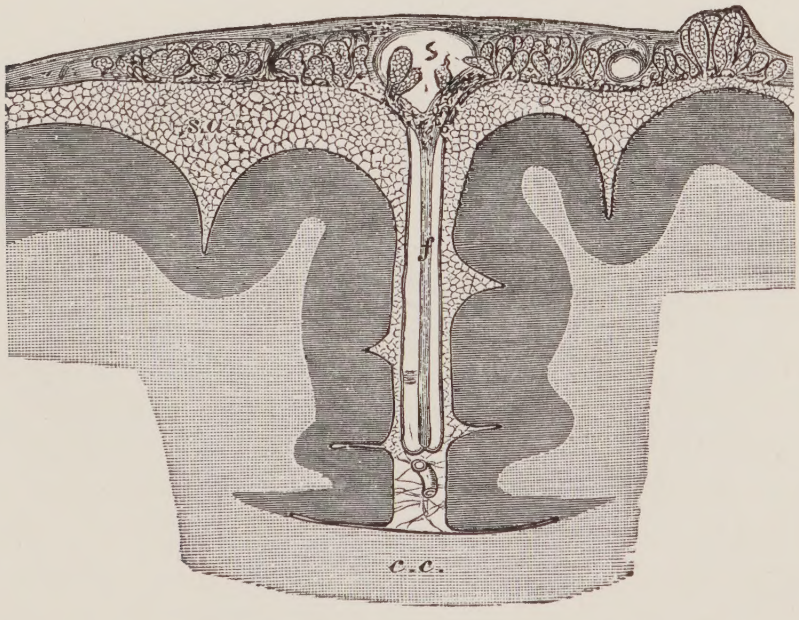
arrested in the usual way (see method of arresting hæmorrhage from the sinuses under chapter on "Treatment"). The dura also sends inwards processes formed by duplications of its inner layer, named the falx cerebri, tentorium cerebelli, and falx cerebelli. In infancy and childhood the membranes of the brain are translucent, and sometimes so transparent as to permit the brain and its pial vessels to be distinctly seen through them.

The connection between the dura mater and the skull increases with age, and becomes much more intimate in chronic inflammatory processes affecting the skull, and in pachy-meningitis; but during acute inflammatory stages the connection between the bone and the dura is much less intimate, and the dura may then be easily separated. The potential interval between the dura mater and the arachnoid, is termed *the subdural space.* It is into this space that pachy-meningitis hæmorrhagica occurs; a considerable effusion may be poured out here, and, owing to its diffusion, may not occasion marked symptoms. On the other hand, blood poured into the subdural space from the rupture of a vessel in the vertex may find its way into the vicinity of the pons, cerebellum, medulla, and the lowest parts of the spine, and cause death by pressure at parts remote from seat of injury. Blood effused into the subdural space, even if it coagulate, is prone to shift its position, and suddenly cause dangerous symptoms. In operations upon the brain, bleeding which passes into the subdural space may diffuse itself widely, and cause dangerous symptoms from the loss of blood, and from the pressure on the fourth ventricle. To obviate such risks in the removal of very vascular intracranial tumours, the author sometimes divides the operation into two stages, the first to secure adhesion of the membranes, thereby obliterating the subdural and subarachnoid spaces, and secondly, proceeding with the tumour removal. Occasionally, if the tumour be superficial, it is extruded by the brain impulse. Pus effused into the subdural space will gravitate in the same way. When it passes into the cerebellar fossa it quickly finds its way into the spinal canal. In intracranial irrigation of abscess cavities, great care must be observed in order to secure the extracranial return of the fluid injected. Were the nozzle of the syringe introduced into the subdural space, the contents of the syringe might be forced into it. When this occurs, the fluid may return quickly; occasionally it is retained, and, when it is so, it has probably carried with it particles of the pus with its pathogenic organ-

isms, washed from the vicinity of the wound to distant parts, so that the poison is further disseminated. In one instance reported to the author by an eye-witness, the intra-membranous retention of several ounces of an antiseptic fluid which was intended to wash out an abscess cavity, was followed by ocular pressure symptoms—widely dilated pupil—on the same side as that into which the fluid was injected, and on the opposite side some cruro-brachial paralysis, both of which however passed off in four days.

Arachnoid.—The arachnoid is situated between the dura and pia maters. It is a thin delicate structure consisting of several layers of interlacing bundles of fibrous tissue, the intervals being filled by expanded cells (Fig. 22). The subarachnoid membranous expansions have a similar structure. It contains few bloodvessels, and no nerves have as yet been demonstrated in it.[1] It is covered on both sides by expanded endothelium. Over the cerebrum it passes from the surface of one convolution to the other, bridging the sulci; and at other parts, over the top and upper part of the sides of the brain, it is closely applied to the

FIG. 22.



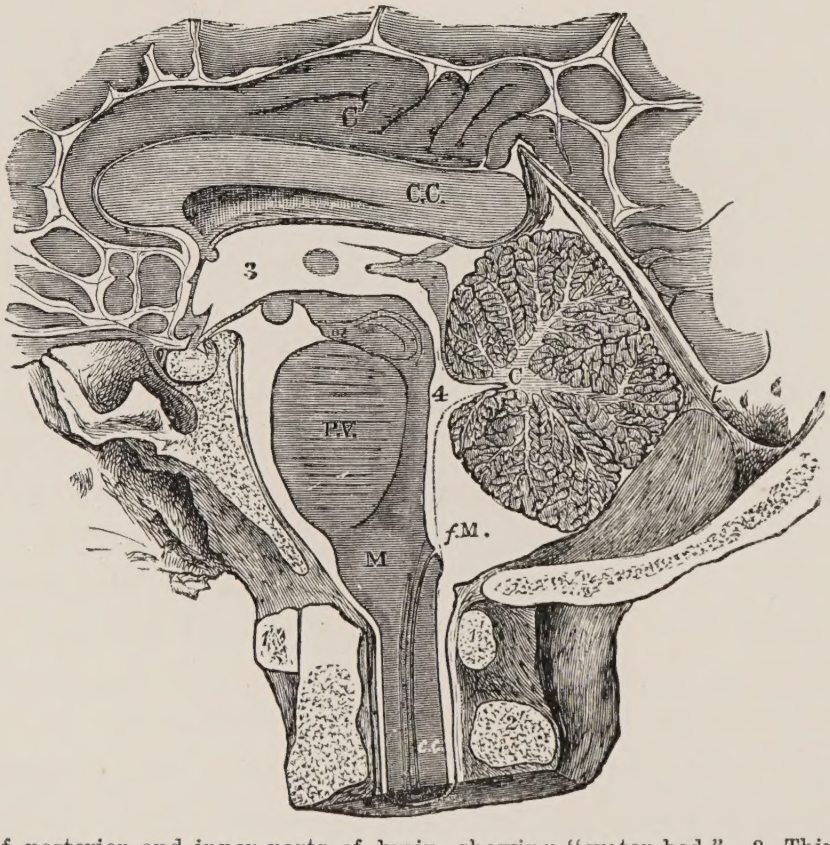
Section of upper part of brain, subarachnoid space, filled by trabeculæ. (From Quain, after Key and Retzius.)

pia mater, so that it is practically impossible to separate the arachnoid as a membrane from the pia.

[1] Volkmann, Bochdalek, and Luschka have detected nerve filaments in the arachnoid of certain ruminants. Bochdalek traces them to the portio minor of the fifth, the facial, and accessory nerves.—Quain, vol. ii. p. 378. [Ed. 1882.]

Between the arachnoid and the pia mater there is an interval called the subarachnoid space, within which the greater part of the cerebro-spinal fluid is contained, a portion of the same fluid being also found in the subdural space. In the subarachnoid space, the larger bloodvessels pass obliquely towards the brain. This space is very unevenly distributed over the brain surface, as at some parts of the convexity, it is scarcely recognizable, while over the posterior two-thirds of the base, there is a wide interval between the arachnoid and the pia mater, extending in front over the medulla, the pons, and the inter-peduncular recess as far forward as the optic nerves; while posteriorly, there is a considerable interval between the cerebellum and the back of the medulla oblongata (Fig. 23). Thus these important parts of the brain rest upon the cerebro-spinal fluid as upon a water bed. The only parts of the base of the brain resting directly upon the membranes covering the bone are those in contact with the orbital plates of the frontal, and those impinging upon the lesser wings

FIG. 23.



Section of posterior and inner parts of brain, showing "water bed." 3, Third ventricle; 4, fourth ventricle; *f*.M, foramen of Magendie; C.C. corpus callosum; C, cerebellum. (Quain, from Key and Retzius.)

of the sphenoid. The subarachnoid space is also of considerable dimensions above the corpus callosum, as the arachnoid at that

point stretches across, just under the falx cerebri. The space is occupied by thin membranous expansions of connective tissue, irregularly interwoven and attached to the pia mater and arachnoid.

There is a communication between the subarachnoid space and the ventricles of the brain, by means of the foramen of Magendie—the opening into the lower part of the fourth ventricle, through the pia matral expansion which closes it. Behind the upper roots of the glosso-pharyngeal nerve, in the pouch-like extension of the pia mater beneath the flocculus, exist two other openings, one on each side. There is likewise a communication between the sheaths of the nerves and the cerebral ventricles, through the intermediary of the subarachnoid space. The nerves, as they make their exit from the brain and spinal cord, receive coverings from the three membranes, the two outer being looser than the inner. This is especially seen in the optic nerve, whose various tunics remain distinct and the spaces between them may be injected—the outer from the subdural, and the inner from the subarachnoid space. The arachnoid sheath is only continued for a short distance over the other nerves, when it becomes fused with the epineurium and perineurium. Fluid injected into the subdural or subarachnoid spaces passes readily along the nerves as far as the limbs, It will thus be seen what an extensive surface is exposed to inflammatory effusion in lepto-meningitis; the whole cerebro-spinal system is exposed and there is a continuity between the subarachnoid space, the perivascular canals of the brain substance, the lymphatic spaces within the nerve sheaths and the ventricles of the brain. Though there is no direct channel between the subdural and subarachnoid spaces, yet fluid can pass through the meshes of the arachnoid into the subdural space. The arachnoid villi (Glandulæ Pacchionii) are seen upon the external surface of the dura mater, pits being formed on the inner side of the calvarium to receive them. They also project on the internal surface of the dura, some of them passing into the longitudinal sinus. These arachnoid villi spring from the arachnoid and penetrate the dura mater. Each villus consists of arachnoid trabeculæ, covered by an arachnoidal expansion, over the surface of which there is a slight covering of the dura mater. The interior of each villus is continuous with the subarachnoid space, while the interval between the dural and arachnoidal covering is continuous with

the subdural space. Fluid can easily be injected into the interior of these villi from the subarachnoid space, and it is found to filter slowly through the inner covering into the subdural space and even into the longitudinal sinus. It is believed, that when the pressure in the subarachnoid space is increased, the cerebro-spinal fluid may escape by means of these arachnoid villi into the longitudinal sinus. May it not also be the case, that when the pressure in the subarachnoid space is diminished, a reverse action may ensue, these villi abstracting the serum from the longitudinal sinus?

Cerebro-spinal fluid.—It is said that the cerebro-spinal fluid equalizes the intracranial pressure to the varying effects of the cerebral blood circulation. When the great nerve centres are swollen by congestion, they are not met by an unyielding structure, but displace some of the cerebro-spinal fluid through the foramen of Magendie. The closure of this foramen may lead to internal hydrocephalus, as was well seen in a case under the writer's care, where the foramen of Magendie was blocked by a cerebellar tumour causing enormous distension of all the ventricles, and consequent thinning of the brain substance. If, during an intracranial operation, the subarachnoid space be opened, excessive expiratory efforts, such as sneezing, coughing, loud expiratory exclamations, cause a welling up of the fluid through the aperture in the cranium though it be situated at the vertex. On each long inspiration the fluid recedes from the aperture, and on each deep expiration it flows out. This may be seen in stertorous breathing. If the skull and membranes be opened at the base the water-bed is encroached on, and much cerebro-spinal fluid may escape. This constitutes a danger of operating at this part, as the relation of parts becomes altered, and the medulla may rest upon the bones to an extent sufficient to interfere with its function. A few hours of occlusion of the aperture will, however, be sufficient to permit the re-accumulation of the fluid sufficiently to prevent any further ill effect. In cases of spina bifida, in which the ventricles of the brain are also enlarged, there is in many instances a free communication existing between the fluid in the thecal membranes and that in the ventricles and in the subarachnoid space. When, therefore, the covering of the spina bifida bursts, the cerebro-spinal fluid wells away from the brain leaving the basal ganglia unsupported. A fatal termination quickly ensues, accompanied by general spasmodic and convulsive

seizures. In certain forms of tubercular lepto-meningitis a great amount of cerebro-spinal fluid accumulates in the subarachnoid space, occasionally limited by adhesions, but more often diffused. This constitutes a form of external hydrocephalus, and when the cranium has already become ossified this excessive accumulation of fluid exercises pressure on the brain which recedes from the osseous walls, becoming reduced in volume, and its function greatly lessened and finally arrested. The quantity of cerebro-spinal fluid which escapes through a fracture of the skull with rupture of membranes is sometimes very great. Bleeding from the nasal septum occasionally relieves congestion of the cerebral veins. May not the serum from the subarachnoid space when superabundant be rapidly eliminated through the same channel? This would be more direct than the elimination, which takes place in much greater quantity through the general system.

The brain may be damaged by contre-coup, though uninjured at the point struck. But the cerebro-spinal fluid greatly modifies and often prevents the damage which otherwise would be apt to ensue from contre-coup; so that when blows are delivered on the vertex, the structures at the base are protected as they lie upon the elastic water-bed. When the force falls upon the occiput, the brain may not be damaged posteriorly, but it is thrown forward within the skull, the water-bed saving the basal portion from being torn against the projecting osseous part of the base. The basal portions of the brain which usually suffer from contre-coup, occasioned by falls on the occiput, are the frontal lobes which are brought violently against the irregular orbital plates, and the anterior basal segments of the temporo-sphenoidal lobe, which impinge against the wing of the sphenoid.

Pia mater.—The pia mater is a delicate highly vascular fibrous membrane, closely investing the brain and spinal cord. The entire cortical surface of the cerebral convolutions is covered by it, each of the sulci being lined with it. It is invaginated at the transverse fissure into the lateral and third ventricles, forming there the velum interpositum or tela choroidea superior and the choroid plexus; and in the posterior wall of the fourth ventricle it forms the tela choroidea inferior and the choroid plexus of that ventricle. It is more closely adherent to the substance of the cerebral convolutions than to those of the cerebellum.

The cerebral pia mater consists of one layer corresponding to the inner of the two layers constituting the pia mater of the cord.

It contains great numbers of bloodvessels, which sub-divide in it before entering the brain. The accompanying photogravure illustrates the vascularity of the pia mater and its intimate connections with the cerebral tissue. Each vessel is enclosed in a perivascular sheath, composed of a denser arrangement of the fibres of the membrane, and the diameter of the lymphatic canal thus formed may be considerably larger than the contained vessel. At its commencement the sheath is loose and funnel-shaped, and can be injected from the subarachnoid cavity, into which the lymphatics of the pia mater open. It is supplied by an extensive network of fine nerve fibres derived from a great many sources,—the sympathetic, oculo-motor, trigeminal, abducens, facial, pneumogastric, glosso-pharyngeal, and accessory nerves. These nerves accompany the vessels into the substance of the brain, and it is supposed they are destined for the vessels alone. The intimate relations of the pia mater and brain, and the manner in which the bloodvessels dip into the cerebral substance, carrying along with them their investment of the pia mater, show that it is impossible to have lepto-meningitis without a degree of encephalitis.

Nerve supply of cerebral membranes.—The *dura mater* is supplied with nerve filaments from the sympathetic, the fourth, the fifth, and the twelfth; while the nerve filaments to the *pia* are derived not only from the sympathetic, but from all the cranial nerves except the first, second, fourth, eighth, and twelfth. Hence severe pressure on the membranes against the bone or inflammation of them occasions widespread pain. Temporo-sphenoidal abscess will press upon all the branches of the fifth at the Gasserian ganglion, and also on the third in its course.

FIG. 24.—CEREBRAL PIA MATER.

The lighter parts represent the membrane which covers the cerebral convolutions, and the darker correspond to the deeply penetrating fringes consisting of thousands of bloodvessels which dip into the sulci and terminate in the brain substance.

A portion of the cerebral pia mater, from an injected specimen in the collection of William Hunter, in the Glasgow Hunterian Museum, photographed by permission of Professor Young.

FIG 24.

CHAPTER II.

PATHOLOGY.

THE pathology of abscess of the brain is so closely interwoven with that of infective meningitis, and the one so frequently accompanies the other, that the pathological aspects of both are dealt with in the same chapter.

In a given case, where a cause of infective inflammation exists on the outside of the skull, from which the interior becomes affected, the alternative, whether meningitis or brain abscess results, depends partly on the anatomical arrangement of the structures, and partly on the intensity and rapidity of the inflammatory action: which again, may be dependent upon the nature of the micro-organism and the virulence of its action. One, or more, of several intracranial conditions—pachy-meningitis, lepto-meningitis, ulceration of the brain, abscess of the brain, and necrosis of the cerebral tissue, besides the involvement of the intracanial sinuses with disintegrating thrombosis—may result therefrom.

Various intracranial lesions which may result from pathogenic and saprophytic causes and the manner in which organisms reach the intracranial structures.

(1.) If the inflammatory process be slow, mild, and distinctly localized, involving a portion of the inner table of the skull, then, an external pachy-meningitis may form, possibly with pus between the dura mater and the bone, producing an extradural abscess.

(2.) Should this condition persist, adhesive inflammation is apt to spread to the inner side of the dura, which may result in a soldering of the inner membranes, the subjacent arachnoid,

and even the pia becoming adherent by the fibrinous meshes of the plastic exudation. Once this occurs general lepto-meningitis is guarded against, in the same way as general pleurisy is prevented from following abscess in the lung by that adhesive inflammation which sometimes solders the visceral and parietal layers of the pleura, only in this case the inflammation spreads from within outwards, in the other, from without inwards. It is possible, however, that in certain brain conditions the inflammation might come from within. This localized soldering of the membranes, once the adhesion has become firm, may act as a barrier to the ingress of fresh infective matter to the general subdural cavity, and to the brain itself.

(3.) After adhesive inflammation has brought about a localized soldering of the soft membranes to the dura, should an increase of the inflammatory action take place sufficient to induce a degenerative inflammation, those membranes with their inflammatory effusion may soften and in the subdural space pus may form, which is prone to be followed by disintegration of the pia and sequent superficial ulceration of the brain tissue. Two conditions may thus result. Should the pia mater remain intact, a subdural abscess may form; should the process extend to purulent softening of the pia and adjacent brain tissue, then cerebral ulceration ensues, the disintegrating products being confined peripherally by the membranes, and by the brain on the inside. In either case the abscess is at first confined to and enclosed laterally within the area of the soldered membranes. Should the abscess enlarge considerably, and the disintegrating process affect the adherent membranes now forming the abscess wall, it is possible that the disintegrating inflammation may spread laterally into the subdural space, or the abscess may burst into it, thereby setting up an acute lepto-meningitis.

(4.) If the cause of the inflammation penetrate through the outer layer of the dura mater into the wide-meshed capillary network of its inner layer before soldering of the membranes has occurred, the whole subdural space is open to invasion, and an acute far-reaching lepto-meningitis is apt to ensue.

(5.) Lepto-meningitis and cerebral abscess may form independently of a visible tract of inflammation spreading inwards from the initial focus of irritation outside the cranial cavity. In such cases, the pathogenic cause has been conveyed through the vascular system by direct extension from the source of

infection to the meninges or brain. This may be done by a thrombosis extending through the veins into the pia or the brain; or the veins may become blocked by a localized disintegrating thrombus, portions of which, containing pathogenic micro-organisms, may be carried inwards by the reversed blood stream. The sinuses and intracranial and intraosseous veins are destitute of valves, and the current of blood in them can be reversed on occlusion occurring at one point. This has been termed a reflex method of propagation. It is clear that when the thrombus passes into and occludes the sinuses, it may rapidly extend into the cerebral or cerebellar veins, and thus convey those organisms into the cerebrum or cerebellum respectively. The veins running into the sigmoid sinus are very numerous, both from the mastoid cells and antrum on the one hand, and from the contiguous parts of the cerebellum on the other. Many of the veins from the temporo-sphenoidal lobe run into the superior petrosal sinus, which also receives numerous veins from the tympanic cavity, and from the neighbouring tegmen tympani. Besides the venous channels, infective matter may be conveyed into the brain substance through the perivascular sheaths of the arteries. It is also possible that some of the cortical branches of the second division of the posterior cerebral artery may become involved, as they lie on the pia mater, covering the base of the middle fossa; pathogenic organisms being conveyed to their immediate vicinity by the minute veins already referred to, or by direct contact with the softened and disintegrating dura and arachnoid over the tegmen tympani. Plastic exudation emanating from an erosion in the tegmen, and accompanying infective processes, is often distinctly prominent in this region. The perivascular sheath of the artery may become affected, and be the channel through which the peccant matter reaches the brain. If it should involve the arterial contents, a localized thrombus may result, of which, if it disintegrates, a portion may be carried from the larger vessel into the terminal capillaries in the white cerebral substance, where it would set up minute infective hæmorrhagic extravasations, round which an abscess might form. In such a case, the abscess of the brain would be formed in the white substance, without the surface of the convolutions showing marked inflammation. It is also possible that a large arterial trunk may become involved, such as the internal carotid, and partial infective thrombosis of that vessel

might lead to infective particles being carried in the arterial circulation to distant parts of the brain, so that abscess might form in other than the temporo-sphenoidal lobes from this method of propagation. The occlusion of a large arterial cerebral artery results in an extensive cerebral necrosis. In a specimen seen by the author, there was an extensive erosion from the middle ear into the carotid canal, the artery being surrounded by granulation tissue, its coats thickened, especially over one side, and its lumen lessened by a soft thrombus. Professor Gairdner and Dr. Barr conjointly reported a case in which there was complete thrombosis of the internal carotid, resulting from intra-tympanic infective inflammation. Inflammation may also spread through the lymphatic vessels to the brain. The perineural sheaths may likewise be the path along which the micro-organisms travel, though one very seldom finds cerebral abscess forming by perineural extension, lepto-meningitis being there the rule. Inflammation may pass along the nerve sheaths from the labyrinth through the internal auditory meatus to the meninges.

Micro-organisms the chief cause of intracranial pyogenic processes.—In the great majority, if not in all cases, the peccant matter, which occasions both acute lepto-meningitis and brain abscess, is organismal. Whether the various forms of intracranial disease have each a distinct specific organism which produces it, and it alone, is for the future to decide. It is, however, probable that the same organism may induce several of these intracranial lesions, the modifying circumstances being, the degree of intensity of its action, its opportunities of access to the intracranial contents afforded by the pathologico-anatomical condition of the parts, and the degree of the restraining force of the living tissues presented by the individual.

The free communication between the naso-pharynx, the tympanum, and the mastoid antrum, is such that diseases due to micro-organisms arising in the first-named part, not infrequently affect the others, while, when the membrana is softened by disease and perforation ensues, organisms have free access to the tympanum directly from without. When the middle ear has become inflamed, and its mucous membrane is converted into granulation tissue and purulent exudation issues from it; then it forms a model incubating chamber for micro-organisms, which find there abundant pabulum, ever freshly poured out, and an even, temperature uniformly favourable for their development.

Once they have penetrated into the mastoid antrum and the adjoining mastoid cells, they are so far removed from the influence of antiseptics applied to the middle through the external ear, that they rest secure and fructify uninterruptedly as long as pabulum is forthcoming.

In the ordinary neglected otorrhœa from middle ear disease swarms of micro-organisms are found both saprophytic and pathogenic. There are monococci, diplococci, streptococci, and staphylococci, occasionally accompanied by bacilli. As might be expected the various cocci and bacilli which affect the naso-pharynx are apt to find their way into the middle ear along the inflamed Eustachian tube. This has been demonstrated by many independent observers. On the other hand, it is also possible for a culture of micro-organisms which have grown in the middle ear to pass along the inflamed Eustachian tube into the naso-pharynx, setting up inflammation there, then if inspired, occasioning infective pneumonia, or if swallowed, infective diarrhœa. In several cases of profuse otorrhœa the discharge from the Eustachian tube has been noticed issuing in drops from the vicinity of the trumpet-shaped orifice in the naso-pharynx though the patients had no expectoration. Evidently they swallowed the discharge, and it was with difficulty they were taught to eject it from the mouth. In one of these cases the patient had profuse foul smelling diarrhœa from enteritis which could have been occasioned by the swallowing of septic matter, though, of course, it could also be explained by infective absorption.

In brain abscess and in suppurative lepto-meningitis organisms of a pathogenic and saprophytic nature are likewise found, and, on several occasions, the same varieties of organisms seen in the discharges from the otitis media have been found in the purulent secretion from the brain. Their variety is such as to prevent a selection of any one as being pre-eminently more potent than others in producing extension of the inflammatory action to the brain. The same varieties of micro-organisms have been seen in the soft thrombi and purulent disintegrations found in thrombosis of the sigmoid sinus. The streptococcus pyogenes and the staphylococcus aureus are frequently found in all these conditions.

The pathogenic organisms found in suppurative lepto-meningitis and brain abscess, as tested both by the microscope and by

careful plate cultivations in the author's cases, have most frequently been the streptococcus pyogenes and the staphylococcus pyogenes aureus. Occasionally the staphylococcus pyogenes albus and citreus have been discovered, but always associated with the streptococcus pyogenes or the staphylococcus aureus. When pus in the intracranial suppurative lesion has been compared with that emanating in the same case from the exterior wound or otorrhœa the same organisms have frequently been found in both. The bacillus pyogenes fœtidus has been found in one case both in the otorrhœa and in the cerebral abscess. In one specimen at least there was present besides the bacillus pyogenes fœtidus the streptococcus pyogenes. In a case of cerebellar abscess accompanied by a large meningeal abscess in the posterior cerebral fossa the pus from the latter showed the diplococcus pneumoniæ of Frænkel (the micrococcus septicus sputigenus). In a case of compound fracture of the skull which became affected with what seemed to be a mild attack of erysipelas, in the suppurative cerebro-spinal lepto-meningitis which followed, the pus contained large quantities of both streptococci pyogenes and staphylococci aurei as well as staphylococci citrei and albi. In a case of suppurative cerebro-spinal lepto-meningitis, the bacillus meningitidis purulentæ of Neumann was found; and in a second case where there was a cerebral abscess, though there may have been slight localized meningitis, a bacillus was found which was either Eberth's bacillus entericus or the bacillus meningitidis purulentæ, but most probably the latter; in several cases, along with more potent pyogenic organisms, there was the bacillus pyocyaneus of Gessard—one of those organisms which impart a greenish colour to the pus, and possibly also to the tissues in its vicinity.

In the cultivation experiments the ordinary saprophytic organisms were rejected. In the pus which is found in the middle ear and antrum these organisms frequently abound along with streptococci and staphylococci. A leptothrix, apparently the leptothrix buccalis, has been seen in the antral pus. On one occasion, in a cerebral abscess containing the staphylococcus aureus and the bacillus pyogenes fœtidus, the tubercle bacillus was likewise seen. In that case there were some sloughs in the abscess of a caseating kind. From the suppurating frontal sinus, besides the streptococcus pyogenes, there was also found the staphylococcus salivarius pyogenes (Biondi).

It must be noted that in these and other cases organisms were found, some of which could not be successfully cultivated, and others which were either unknown bacilli or were not recognized.

Nor is it merely in the purulent otorrhœa that organisms are found. The cholesteatomata which form in the cavity of the tympanum are filled with them, and as these masses of epithelial debris and inspissated pus resist the action of antiseptic washings of the middle ear, they harbour and preserve germs sufficient to infect new pabulum as it may be secreted.

The virulence of a discharge cannot be measured by its odour. Nearly odourless otorrhœa may contain pathogenic micrococci, and some of the most serious intracranial inflammatory lesions ensue in the presence of an odourless otitis media. It is well, therefore, in estimating the gravity of an otorrhœa, that the pus from the middle ear should be stained and examined microscopically and by cultivations. Though cerebral abscess often does occur as a result of non-fœtid otorrhœa, yet many such intracranial lesions arise in the presence of otorrhœa with intense fœtor. It does not, however, follow that the bacillus producing this fœtor is the cause of the intracranial lesion, as many bacilli which do produce fœtor have been found by Röhrer to be non-pathogenic. There are some bacilli, however, which possess the power of disintegrating the albuminous molecule with the development of fœtid gaseous products, and at the same time causing putrefactive fermentation with pyogenic results. Such, according to Passet, is the *bacillus pyogenes fœtidus*, which, when introduced into the subcutaneous tissue, does produce suppuration (Dr. Flügge, transl. by Watson Cheyne, p. 375).

The mere chronicity of an otorrhœa, without extension of serious disease, is no guarantee to the individual of future immunity. Patients so affected are at any time liable to the rapid onset of dangerous sequelæ. This may arise from fresh inoculation of the discharges in the middle ear, or from the exposure of fresh areas of the membranes, brought about by progressive disintegration and the implication of these fresh areas in the pathogenic process.

Though acute attacks of infective inflammation of the skull, such as from compound fractures, frequently lead to intracranial extension of the pathogenic process, yet primary acute attacks of otitis media are rarely followed by extension of the pathogenic process to the brain or its membranes. This immunity of the cerebral

structures from implication in presence of primary though severe attacks of otitis media, is accounted for by the fact, that the bone and the mucous membrane of the middle ear are still intact, and present a barrier to infection travelling inwards. The virulence of the inflammation might, however, even under such circumstances produce thrombosis of a sinus, or give rise to other intracranial complication. Thus, diphtheria has extended from the pharynx into the middle ear, and there set up intense inflammatory action with ensuing thrombosis of the veins, which extended into the sinus and finally occluded it. In the same way, cocci have been found to enter the jugular vein, through a purulent canal extending from the original diphtheritic affection of the tonsil, and afterwards set up suppurative inflammation of the joints.[1] That the paths of entrance for micro-organisms into the intracranial tissues are ample may be admitted when it is known that lung pigment has been found in a cerebral abscess[2] secondary to pulmonary suppuration, and that the oidium albicans which affected the mouth and pharynx, penetrating the epithelial layer, has entered the blood, and has given rise to abscess of the brain, the oidium being found in the abscesses.[3] There are two such cases reported—one in a child, the other in an adult.

THE KIND OF INJURIES AND PATHOLOGICAL CONDITIONS LEADING TO INTRACRANIAL DISEASE.

(1.) *Punctured wounds penetrating into the bone.*—The consideration of the kind of injuries and pathological conditions which are prone to give rise to these various intracranial lesions is important. The chief among the injuries in this respect are punctured wounds which penetrate the soft tissues of the scalp, expose and bruise the skull over a small area, and inoculate the bone with infective matter engrained into its surface. Infective thrombosis follows in the diploic vessels, which extends along the perivascular sheaths to the brain. When superficial suppuration does arise in punctured wounds, the free escape of pus is prevented by the firmly bound periosteum, with its minute punctured aperture. Such wounds are additionally dangerous, as externally they appear so small and insignificant that they are often treated lightly and

[1] Dr. Flügge, p. 195. Eng. Trans. Mr. Cheyne—original art. in *Berl. Klin. Woch.* 1884. No. 44.

[2] Böttcher, *Petersb. Med. Zeitschrift*, 1869, and *Virchow's Jahresbericht*, 1869, ii. 51. Gower's *Diseases of the Nervous System*, vol. ii., p. 442.

[3] Zenker, *Bericht der Gesellsch. f. Nat. u. Heilk.*—Dresden, 1861, p. 62. Ribbert, *Berl. Kl. Wochenschr.* 1879, p. 617. Gower's *op. cit.*, p. 443.

may heal superficially with great facility—occasionally within forty-eight hours. The injured person often goes about his ordinary duties and believes himself well. Days or weeks subsequently distinct cerebral disturbance manifests itself. Occasionally, the period elapsing between the injury and the exhibition of the intracranial symptoms is much prolonged. It may happen, in addition to what has been described in the original injury, that the dura mater has been detached from the skull over a small area, and blood has accumulated between the bone and the dura. When this occurs, the infective matter is apt to find a nidus for its growth, and subsequent inflammation occasions extradural abscess.

(2.) *Punctured fractures* are also very liable to induce intracranial disturbance, as the infective matter is here introduced, not only between the fractured surfaces, but also on the intracranial side, or even into the brain itself. Lepto-meningitis is not infrequent after such injuries when left to pursue their own course. Cerebral abscess follows when the brain is punctured and the pathogenic organisms are directly deposited. Owing to the very small external wound often associated with punctured fractures of the skull, they are apt to be overlooked; when discovered, no external ablution will remove the infective matter ingrained into the edges of the fracture and deposited in the soft intracranial tissues.

Case I. In a case which occurred in 1879, two punctured fractures on the vertex of a young man were produced by a blow with a pair of tongs. The external wounds were insignificant, but the fractures exposed the dura, and one of them penetrated to the inner membranes, as indicated by the escape of a small quantity of cerebro-spinal fluid. Three days after the injury (during which, though not at work, he was going about out of doors) he had a rigor followed by high temperature, and lepto-meningitis, from which he died on the fifth day after the injury. The post-mortem showed that his whole cerebro-spinal system was affected with lepto-meningitis, spreading from the punctured fracture which had penetrated the dura. The whole of the cerebral pia mater was deeply injected, many of the pial vessels were thrombosed, especially about the point of penetration. At this part, and for a couple of inches round it, there was a small purulent exudation.

Though this case was not under the author's care, the facts are known to him personally, as he was present at the post-mortem.

(3.) *Compound fractures* with large open wounds are much less liable to produce cerebral abscess than punctured fractures, be-

cause of the free escape afforded for discharges, and from the fact that they present a much more serious aspect, and are treated accordingly. When intracranial abscess does occur in connection with them it is generally seated in their immediate vicinity, often extradural; and when the brain is involved, the abscess is generally superficial surrounding the wound in the cerebral substance, though the white matter is usually implicated. When there has been injury to the brain from *contre-coup*, abscess may form at the point opposite the part struck. Traumatic abscess is usually single.

When intracranial suppuration arises, it does so from pyogenic organisms which have been permitted to remain in the tissues. The fragments of bone are often ingrained with dirt, which requires to be removed with the chisel, and they have to be very carefully scrutinized, as the debris is apt to be overlooked. Between the edges of linear fractures, or the margins of the compound comminuted fractures, there is often lodged infective debris, which is apt to be concealed, as the portion of bone which forms one of their margins has sprung back to its normal level on the cessation of the impact. By levering the margins slightly apart with the edge of a chisel, hairs and foreign matter may be seen and removed. Again, such matter may be driven directly into the brain substance by one of the osseous fragments, or by the foreign body producing the lesion, after the withdrawal of which the brain tissue remains inoculated; so that it may be a matter of great difficulty, without interfering unduly with the brain substance, to be sure that all infective matter has been absolutely removed. Though this be true, yet, in a very large experience of compound fractures of the skull, often accompanied by rupture of the dura mater and penetration of the brain substance, the author has met with only a single instance of cerebral abscess in cases which have come *de novo* under his personal supervision.

CASE II. *Extradural abscess, with pachy-meningitis, resulting from infective compound fracture of skull. Coming under observation on fifth week of illness. Duration of illness, six weeks. Recovery.*

J. W., aged 28, was admitted into the Glasgow Royal Infirmary on 9th October, 1883, suffering from symptoms of intracranial suppuration. He was a strong, otherwise healthy man.

History.—*Heredity.*—No hereditary taint. *Personal.*—Seven weeks prior to admission he fell down the slope of a quarry, his head coming in contact with a projecting stone. In this way he received a small wound in the upper and back part of the head, which gave him little trouble and was soon healed.

Present Illness.—About three weeks after the accident he began to experience a dull aching pain over the vertex, especially on the left side. This pain sometimes extended from the brow to the back of the head, and though generally of a dull aching character it occasionally became more acute and even lancinating. He soon afterwards began to suffer from great mental depression, and complained of a sensation of weight in his head. These symptoms continued until his admission into hospital, four weeks from their onset.

After being in the ward for a few days, besides the persistence of the symptoms just indicated, he was seen to be irritable, restless, and sleepless. He preferred, however, to lie in bed, as he felt giddy when he moved about. The pain from which he suffered was referred to the vertex and brow, more than to the vicinity of the cicatrix. The cicatrix was situated to the left of the middle line at the posterior extremity of the parietal bone. On careful palpation there was a slight tumidity discerned about the cicatrix—Pott's puffy tumour. His temperature was slightly over 100° F. Pulse about normal. He had had no rigors, though he experienced a sensation of cold.

From these symptoms and appearances it was concluded he was suffering from suppurative pachy-meningitis, most probably connected with and for the most part localized at the seat of previous injury. It was resolved to elevate the bone in the vicinity of the cicatrix for the exit of pus which was believed to exist, probably on the outside of the dura mater.

Operation (11th October, 1883).—Patient having been anæsthetized, an incision was made across the cicatrix, situated near the junction of the parietal and occipital bones, about an inch from the middle line. The periosteum was thick and somewhat infiltrated with plastic exudation. The cranial bones were drier than usual, and slightly inflamed. A stellate fracture of the skull was discovered, accompanied by a slight depression of the external table. While a disc of bone was in process of removal from this point, the external table came away in two portions, exposing a much depressed and comminuted internal table, which seemed to have been partially separated from the external table. The internal table was much more extensively fractured than the external. Six pieces of the former, which were depressed and partially adherent to the dura mater, were removed, and by this means exit was given to about four drachms of pus. At the centre of the affected area the dura mater was covered by a thick layer of granulation tissue, while at its circumference this membrane was flocculent. The brain pulsations were distinct. The thickened layer of granulation tissue attached to the dura mater was removed. The parts were then washed with an antiseptic solution, and the two portions of the external table of the skull, which had been meantime preserved in an aseptic sponge, were now divided into several

portions and re-implanted, space being left for drainage. Those portions of bone were drier than normal, and slightly inflamed ; it was therefore doubtful whether they would live. The wound was then stitched, a decalcified drainage tube introduced, and an absorbent dressing applied.

Subsequent Progress.—A fortnight afterwards his wound was healed. His former mental depression and painful sensations had entirely disappeared. His temperature had never been above normal. A month after the operation he was dismissed well.

He visited the ward three months, and also twelve months after dismissal to report himself. He had been regularly at work since going out, and felt in excellent health. There were no uncomfortable sensations about the head. The re-implanted bone was firm, though there was a slight depression, the external table only having been re-implanted, and probably a little subsidence from the external level of the skull had taken place.

CASE III. *Extradural abscess with pachy-meningitis occurring at the seat of a compound fracture of the frontal sinuses, with injury of the internal table of the skull, received about three months previously. Removal of necrosed bone and evacuation of the abscess. Recovery.*

A. M'P., aged 33 years, labourer, was admitted into Royal Infirmary on 5th January, 1885, suffering from cerebral symptoms pointing to pachy-meningitis and intracranial suppuration.

History.—He was a strong healthy man. Three months previously he had received a blow on the head from a piece of wood ; a compound fracture with depression resulted, which, not having been rendered aseptic, consequently suppurated, and a small piece of bone in a state of necrosis was then removed from the outer table.

Condition on Admission.—When admitted he suffered from dull pain in the front of his head, and had violent lancinating pains shooting from the forehead backwards, and occasionally flashes of light before his eyes. He felt sick, frequently vomited, and was troubled with restlessness and insomnia. His temperature varied from 101°-102·4° F. On the head being examined, there was a soft tumid cicatrix quite healed externally situated over the centre of the brow. When this was incised it was seen to be intimately adherent to a mass of connective tissue, which partly filled the frontal sinuses. There was a complete gap in the external table exposing the sinuses, and the internal table was found to be depressed below the level of the posterior wall of the sinuses and to be in part necrosed.

Operation.—As the symptoms pointed to intracranial abscess along with pachy-meningitis, the skull was trephined near the middle line. When the disc of the internal table was elevated, a quantity of fœtid pus escaped—probably about three drachms—exposing a flocculent dura mater which had been in great part covered with granulation tissue. A few small portions of bone which had become necrosed were removed. The granulation tissue was scooped out, along with the infiltrated pus.

Subsequent Progress.—After the operation all his disagreeable sensations disappeared, his temperature fell on the third day to normal, and his wound healed quickly, leaving, however, a depressed cicatrix. He visited the wards on several occasions after his dismissal, and on each he reported himself as healthy and regularly at work, never having experienced any inconvenience from his head.

(4.) *Infective injury to skull without fracture.*—In some instances, however, without any fracture of the cranium, but where the outer table of the skull has been exposed and ingrained with dirt containing pathogenic organisms, abscess of the brain is a sequence. The wound in the soft tissues frequently heals, and everything looks well externally. After the lapse of a variable interval, generally weeks, symptoms of cerebral disturbance manifest themselves. At the same time the tissues about the cicatrix become swollen, sometimes puffy ("Pott's puffy tumour"), and occasionally, on reopening the wound, a few drops of pus are found on the outside of the skull, and frequently the cicatrix is non-adherent to the bone, the external table of which is in a low state of vitality, and may even be necrosed. On the interior of the skull—extradurally—there are also signs of inflammatory action and usually a few drops of pus. Frequently there is a distinct cerebral abscess situated in the white matter of the brain, which may either be in direct continuity with the injured dura through a zone of inflammation, or may be isolated, fairly healthy cerebral tissue intervening between the seat of injury and the abscess. The paths of communication between the infective injury and the abscess in the latter case will then be vascular or perivascular as above described.[1]

(5.) *Infective necrosis of the skull.*—Instances do occur where large areas of the skull, without being fractured, have become denuded of the periosteum, deep furrows having been ploughed up all over its surface and infective matter scratched into them, the soft tissues of the scalp being at the same time crushed, infiltrated with dirt, and having in consequence become gangrenous. The dura is sometimes stripped from the bone by concussion, and when this occurs along with extensive periosteal separation, necrosis results. In such cases an extradural abscess forms, and, if not attended to by prompt multiple trephinings, may lead to cerebral abscess.

(6.) *Pathogenic affections of the face and scalp.*—Acute pathogenic affections of the face and scalp are apt to produce lepto-meningitis,

[1] See case of infective bruising of the tissue without visible wound, Case LXIV.

with or without sinus thrombosis, but they seldom lead to cerebral abscess though the lepto-meningitis induced is frequently suppurative. Among these affections, erysipelas of the face or scalp frequently sets up thrombosis of the veins, which may extend into the cranium, carrying the streptococci into the membranes and even occasionally into the brain itself. This is not an uncommon form of fatal lepto-meningitis met with in elderly people.[1]

INFLAMMATION OF THE MIDDLE EAR RELATIVE TO INTRACRANIAL INFLAMMATION.—The condition of the middle ear, viewed relatively to intracranial inflammation, is of great importance, as it is in the tympanum and antrum that the majority of the pathogenic processes are generated which afterwards spread intracranially and affect the brain and its membranes. As the mucous membrane of the middle ear is directly continuous with that of the pharynx, it often participates in the inflammatory action of the latter. Thus after measles and scarlet fever, when the throat has been affected, the middle ear is frequently the seat of acute inflammation and suppuration as a sequel. Diphtheria affecting the throat has also been known to extend to the middle ear by way of the Eustachian tube.[2]

Acute processes seldom spread into the interior of the skull.—These acute processes seldom spread into the interior of the skull, because the mucous membrane of the middle ear is still intact, and the underlying bone is unaffected. These structures, therefore, present a barrier to the ingress of pathogenic elements. In such cases the lymphatics are active as may be seen from the enlarged glands, both cervical and post-auricular. Occasionally, however, when the inflammatory action is sufficiently acute to give rise to thrombosis in the vessels of the mucous membrane, the thrombus spreads into the vessels of the bone, and may even reach the intracranial membranes and sinuses.[3]

The existence of purulent disease of middle ear without rupture of the tympanic membrane.—The fact that extensive purulent disease of the middle ear may exist without any discharge

[1] For infective involvement of frontal sinuses see Cases LXI.-LXIII.

[2] One such instance occurring in the practice of the author is reported under thrombosis of the sigmoid sinus; other cases of diphtheria extending to the middle ear from the throat are reported by Dr. Katz, of Berlin, *Deutsche Medicinische Wochenschrift*, 1887, No. 48.

[3] A striking instance of an acute disease spreading from the throat to the middle ear is given under thrombosis of the sigmoid sinus. The disease spread intracranially, and affected the sinus and internal jugular.

escaping from the external auditory meatus, and without perforation of the membrane, must not be overlooked while investigating cases of intracranial inflammation sequent to acute disease of the tympanum.[1]

It is, however, the chronic affections of the middle ear which are most prone to permit extension of pathogenic processes to the intracranial structures. When the mucous membrane of the general cavity of the tympanum is affected with chronic inflammation the deeper structures are very apt to become involved. The underlying bone, the vascular supply of which is in many parts directly continuous with that of the mucous membrane, gradually participates in the degeneration. Molecular disintegration occurs, and, when established, the source of irritation and weakness still persisting, it extends into the more inaccessible recesses of the middle ear, the mastoid antrum, and cells. The mucous membrane of all these parts is destroyed. Granulation masses form and project into the various cavities, further blocking the already narrowed egress for discharge. Frequently the mastoid antrum is greatly enlarged by the destruction of the partitions between the mastoid cells, and occasionally the whole mastoid may be thrown into one wide, irregularly formed cavity, free communication being formed between the tympanum and the tip of the mastoid process.

It may be that the cavity of the middle ear, though enlarged at the expense of its osseous walls, may yet have its internal capacity diminished owing to the encroachment of these granulation masses. It also happens in some chronic cases that the inflammation sets up an osteoplastic formation in the neighbouring bone, which results in eburnating sclerosis and an actual obliteration of many of the mastoid cells, a narrowing of the antrum, and an encroachment on the lumen of the middle ear itself. This osteoplastic process may not affect the bone uniformly, as, while it is occurring at one part, disintegration may be advancing in another. Thus, while the mastoid is eburnated and very dense, the tegmen tympani may be affected by molecular

[1] A case of extensive disease of the middle ear (the fresh specimen of which was sent me by Dr. Erskine for examination), with thrombosis of the sigmoid sinus and osseous erosion without perforation of the membrana tympani, is reported by Dr. Erskine in *British Medical Journal*, 20th September, 1890, p. 678, vol. ii. Habermann ("Mittheilung über Tuberculose des Gehörorgans," *Prager Med. Wochenschrift*, No. 6, 1885, also *Prager Zeitschrift für Heilkunde*, band vi.) found in one instance tuberculosis in the middle ear and mastoid cells, while the membrana tympani was intact.

disintegration. When a fresh pathogenic process is set up in the middle ear of such a case it is all the more apt to find its way inwards, owing to the resistance met with externally in the dense walls by which it is surrounded.

The relative frequency with which the different parts of the temporal bone are attacked by caries and necrosis, though subject to variation, is as follows:—First in frequency is the mastoid process; second, the roof of the tympanum and antrum; third, the sigmoid groove; fourth, the posterior wall of the external auditory canal; fifth, the tympanic floor, with the posterior wall of the carotid canal; and sixth, the petrous portion, including the structures constituting the inner ear.[1]

Anatomical arrangement of temporal bone in children relative to disease of middle ear and mastoid.—The various parts of the temporal bone are not united in young children, and in the infant the component parts are held together by a fibrous matrix, and consequently secretions from disease of the middle ear find a comparatively easy egress.

Sub-periosteal mastoid abscess.—In this way sub-periosteal mastoid abscesses are apt to form, some of which reach a large size and present a very formidable aspect. When such cases are treated promptly they seldom induce abscess of the brain or meningitis, though there is sometimes necrosis of portions of the mastoid, and in one neglected case met with by the author there was necrosis of the whole squamous and mastoid portions of the temporal, with extensive meningitis and abscess in the temporo-sphenoidal lobe.

Acute periostitis of mastoid and squamous bone rapidly passing on to abscess, is somewhat uncommon. When it occurs, it is apt to occasion an amount of alarm on the part of the patient and friends disproportionate to its gravity, as when uncomplicated and promptly treated, though a severe affection, it is seldom fatal.

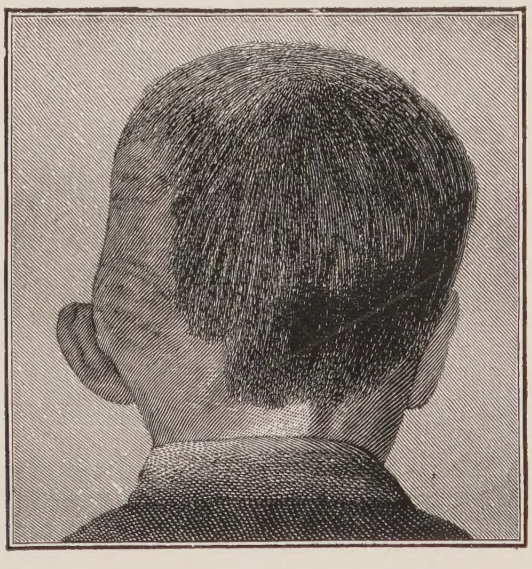
Its origin, in the majority of cases, is in infective otitis media, which has invaded the antrum, and possibly the mastoid cells. In children, the middle ear and antrum are alone

[1] It has been held that sclerosis in the vicinity of the disintegrating osseous tissue forms a natural barrier against the extension of disease to the brain and its membranes. But it does not always form between the disease and the cerebral or cerebellar fossa, as in many instances the sclerosis is most dense on the exterior of the bone while molecular disintegration proceeds toward the brain and its membranes.

FIG. 25.—ACUTE SUB-PERIOSTEAL MASTOID ABSCESS.

Droop of right ear as seen in acute sub-periosteal mastoid abscess (Case IV.).

FIG. 26.—ACUTE SUB-PERIOSTEAL SQUAMO-MASTOID ABSCESS.



Downward, outward, and forward displacement of upper part of left ear in acute sub-periosteal squamo-mastoid abscess. Back view (Case VI.).

To face page 64.

affected, the infective matter issuing in the interspace between the plate of the squamous which covers the antrum and the mastoid. As the periosteum is more easily separated upwards over the squamous portion than downwards over the tip of the mastoid, it is in the upward direction that the pus generally passes. Consequently, the abscess forms more prominently above and behind the ear. The periosteal abscess is but a part of a more deeply seated disease.

It generally occurs in persons subject to chronic otitis media who are in an impoverished condition. At the commencement of the attack, otalgia usually sets in coincident with the cessation of the otorrhœa. The pain often extends until the whole side of the head becomes involved. Vomiting, want of appetite, increase of temperature, and other manifestations, such as accompany periostitis in other parts of the body present themselves. A few days, occasionally twenty-four hours, from the onset of the acute symptoms, a swelling over the upper part of the mastoid process shows itself, which rapidly increases until the whole of the external aspect of the temporal bone becomes stripped of its periosteum, and the upper part of the ear is lifted outwards, and appears at a lower level than its neighbour, giving the head a lop-sided appearance. The boundaries of the abscess are generally at the sutures, where the periosteum dips between the bones.

Acute periostitis of the mastoid and squamous of temporal may however supervene on cerebral abscess, though this is a rare complication of the latter.[1] Chronic periostitis is a frequent accompaniment of sinus thrombosis, and acute periostitis might sometimes attend it.

Evacuation of the contents of the sub-periosteal abscess speedily improves the condition of the patient, provided there is no intracranial complication.

CASES IV., V., VI. *Précis of three cases of acute sub-periosteal abscess involving mastoid and squamous portion of temporal.*

Though acute sub-periosteal abscess involving not only the mastoid but also the squamous portion of the temporal is not very common, three cases occurred and were admitted under author's care, in as many months, into

[1] On one occasion, the author had the opportunity of seeing a case of abscess in the temporo-sphenoidal lobe in a girl aged 14 years, in which there was, as a complication, a large sub-periosteal abscess, occupying the whole external surface of the temporal bone.

the Children's Hospital, in May, June, and July, 1890. A brief note of these is as follows :—

Case IV. **C. C.**, 7 years old, a weak child with a tubercular history, had otorrhœa for about a year, when severe pain in the right ear and forehead occurred. Some days after, swelling arose behind and then above the right ear, which steadily increased until his admission eight days from the onset of his illness. The appearance of his temporal region on admission was photographed. His temperature was between 100° and 101° F.

His right ear hung down, the upper part being pushed outwards. The whole ear appeared to be on a lower level than its neighbour. The direction of the external auditory canal was altered. The membrane was gone, and granulation tissue presented at the orifice of the middle ear. On operation the whole external surface of the temporal bone was found bare, the periosteum elevated, and about four ounces of pus were removed from under it. The middle ear and antrum were likewise cleared out, as they were filled with granulation tissue and foul-smelling pus. He made an uninterrupted recovery.

Case V. **J. C.**, æt. 8 years, was admitted June, 1890, with a history of ear disease of two months' duration, though it probably was of an earlier date. Purulent otorrhœa had existed for six weeks, during which there was considerable otalgia and cephalalgia. Four days prior to admission the mastoid region began to swell. He vomited once. Two days before admission he fainted, and was unconscious for ten minutes. There were no rigors reported. The membrana tympani had been perforated. Fœtid otorrhœa was present in small quantity. The temperature was about 101° F. The photograph shows the condition of the right ear, and the swelling formed by the abscess.

The abscess occupied the greater part of the external surface of the temporal region, though the swelling due to œdema of the tissue formed a considerable margin in the periphery. The abscess was freely evacuated, and pus amounting to $3\frac{1}{2}$ ounces was found in the cellular tissue, besides what was under the periosteum. The patient was soon well.

Case VI. **A. R.**, 3 years of age, was admitted in July, 1890. She was a weak child, and had had otorrhœa when two weeks old, which had continued till four days before admission, when she was seized with otalgia and cephalalgia. Her temperature rose to 101°, and swelling formed behind ear, and rapidly spread over top of ear and side of head. The appearance is seen in photograph.

The whole of the external surface of the temporal bone was denuded of periosteum, saving the tip of mastoid. Three ounces of pus were evacuated, after which the mastoid antrum and middle ear were exposed and cleansed. Patient quickly recovered.

These three cases are illustrative of nine others under personal observation which pursued a somewhat similar course.

While the communication with the exterior of the skull is greater in young children the vascular connections between the

FIG. 27.—ACUTE SUB-PERIOSTEAL SQUAMO-MASTOID ABSCESS.

Front view of appearances presented in acute sub-periosteal squamo-mastoid abscess. The forward displacement of the ear is well illustrated (Case V.).

FIG. 28.—ACUTE SUB-PERIOSTEAL SQUAMO-MASTOID ABSCESS.

Appearances presented in acute sub-periosteal squamo-mastoid abscess of large size (Case VI.).

To face page 66.

dura and the bone are also much more free than in adult life, and meningitis is apt to ensue from pathogenic states of the tympanum.

The mode of infective extension from the tympanum.—Caries in the tympanic cavity does not usually affect the bone equally all round, but extends in certain directions. When it extends through the antrum and involves the mastoid cells the inner walls of some of which are very thin and contiguous to the sigmoid groove, the latter is apt to be involved and exposed. When it attacks the roof of the middle ear, the tegmen, though a dense plate of bone, yet is thin, and when once ulceration does secure a hold upon it, perforation of the middle fossa of the skull frequently results. When the tegmen is in this thinned brittle condition it is very apt to break on slight concussion, produced by blows on the head, or by vibration of the hammer and chisel when used in opening sclerosed mastoids. When the molecular disintegration of the middle ear proceeds inwards and forwards, the whole of the interior of the petrous bone may become, in extreme cases, hollowed out, leaving a shell externally. The labyrinth—the cochlea, and the semicircular canals—being encased in harder bone, often resist the disintegrating process, though they also are occasionally invaded. Such extensive destruction in this direction is uncommon, and is met with chiefly as a tubercular disintegration. Occasionally both petrous bones are similarly affected. On post-mortem examination the tubercular deposition is sometimes found to be continuous from one middle ear to the other through the petrous bone, the basi-sphenoid and basi-occipital being likewise extensively involved. Tubercular meningitis is sometimes a sequel of such changes. On the other hand, when the petrous bone is the seat of extensive pathogenic processes, suppurative lepto-meningitis of the basal ganglia and cerebellum is apt to occur: though this may even ensue when the destruction is much less, but the internal auditory meatus is exposed, the infective inflammation passing along the nerve sheaths of the facial and auditory nerves.[1] Lepto-meningites of this nature occur, which are erroneously ascribed to other causes, as the otitis media has not been a prominent feature. The extent of the latter cannot be measured by the amount of purulent discharge issuing

[1] Infective involvement of the facial and auditory nerve sheaths is much more prone to end in suppurative lepto-meningitis than in abscess in the cerebellar fossa.

from the external auditory meatus, as very extensive disintegration may proceed in the middle ear and its recesses by a process closely resembling "caries sicca," though there is generally a slight aural discharge, an oozing of muco-purulent matter. The osseous ulceration may either extend into the carotid canal (though this is fortunately rare, as when it occurs it may lead to thrombosis of the carotid artery), or it may erode the roof of the jugular fossa.

Most frequent seats of osseous perforation.—The most frequent seats of perforation from middle ear disease are the tegmen over the tympanum or antrum and the sigmoid groove. When an abscess follows the former it is situated in the temporo-sphenoidal lobe, and when it follows the latter the abscess is generally located in the cerebellum. Before actual erosion occurs, the bone forming the tegmen has generally become thinned and discoloured, and in children and young adults the minute veins passing through the petro-squamosal suture are often thrombosed, and carry pathogenic organisms with them into the dura mater. A like discolouration—generally dark—occurs in the sigmoid groove before perforation of the groove takes place. Here also there may be extensive thrombosis of the minute veins between the sinus and the mastoid cells. The tympanic ossicles frequently participate in the ulcerative action, and present eroded and discoloured surfaces.

The discoloration which affects the bone is generally of a black shade, but when acute infective processes are set up, the ulcerated bone is often of a deep greenish hue. In such cases the veins implicated by infective thrombosis are not only stained dark green themselves, but the tissues on which they lie are similarly discoloured. Thus the course of the sigmoid sinus is often marked on the cerebellum by a dark green impression, of a shade similar to the dark staining in the sigmoid groove. The staining and the chemical erosion produced upon the tissues may be due to the bacillus pyocyaneus of Gessard, which imparts the greenish hue to the culture gelatine for a considerable distance beyond the limits of the growth of the organism, or to other chromogenic organisms.

Some consequences of osseous erosion and necrosis.—When the dura mater has been exposed by osseous erosion it throws out in advance of this lesion a mass of granulation tissue from its external surface, which not only offers obstruction to the further inroads of the pathogenic process, but also assists in

the absorption of the disintegrating bone. Such masses of granulation tissue, projecting into the middle ear from the dura through the erosion in the tegmen, have been mistaken for so-called "aural polypi" and have been abrupted or snared, and not infrequently serious intracranial inflammatory action has followed, apparently as the consequence. Similar granulation masses are to be found issuing from the layer of dura forming the wall of the sigmoid sinus through the sigmoid groove into the mastoid cells. These masses of granulation tissue are usually surrounded with pus, a portion of which is pent up intracranially, forming an extradural abscess, the contents of which ooze out if the neck of the granulation mass, as it issues through the erosion, be pressed gently aside. Occasionally, however, the granulation tissue may be accompanied by very little pus, the erosion taking place with all the greater insidiousness. In not a few cases there exists a small necrosis of the bone in the tegmen tympani or sigmoid groove. The extradural abscess forming on the inner side of it.

The tegmen in the neighbourhood of the petro-squamosal suture, and the knee of the sigmoid groove, as well as the portion of the sigmoid extending lower down, are frequent seats of those small necrosed portions of bone.

CASE VII. *Case of extradural abscess over right tegmen tympani with lepto-meningitis accompanied by phlebitis of sigmoid sinus. Operation. Recovery. Coming under observation on twelfth day of his illness. Duration of illness fifteen days.*

J. M'B., aged 36, admitted into Royal Infirmary, 5th January, 1892, on twelfth day of his illness, suffering from extradural abscess in middle fossa, and phlebitis of sigmoid sinus, due to chronic otitis media of right side.

History.—*Heredity.*—Nothing definite in family history. *Personal.*—Has been in fairly good health all his life, although he had the ordinary diseases of early childhood.

Twenty years ago he became chilled and had severe pain in the right ear, which lasted a few days, and was relieved after a free purulent discharge had issued from that ear. Since then the discharge has continued, and he has been more or less deaf. He has, however, been able to continue his work.

Twelve days ago he was seized with pain in the right ear, which was so severe as to cause him to go to bed. This was accompanied by giddiness, a confused feeling, and a cessation of the discharge. On the fourth day he was very sick and vomited frequently, and had a continuous sensation of cold. He thought there was a little swelling behind the ear at this time, and he has perceived a bad taste in his mouth for the last few days. Yesterday he had a severe rigor.

Condition on Admission.—Patient was very prostrate on admission; he had a greyish colour of face, and lay on his right side with his hand covering the right ear, and evidently suffering much pain. He was perfectly clear and intelligent, but hyper-sensitive to external impressions; slight touch, vibration, or noise caused him considerable annoyance. His head was slightly retracted, he had pain at the back of the ear, and down the course of the internal jugular. The upper part of the course of the internal jugular, behind the sterno-mastoid muscle, was slightly swollen, hard and surrounded by enlarged glands.

No paralysis, no in-coordination.

Eyes.—Pupils equal and reacted to light. Fundi had slight swelling of inner margins of both discs more especially of the upper parts. The margins were blurred, vessels of natural size.

Ears.—Discharge from the right ear of fœtid pus, membrane gone, granulation tissue filling middle ear. Hearing on right side absent as tested both by bone and aerial conduction. Left ear normal.

Pulse rate was slightly higher than normal, 70 to 84. Temperature high, 102·2° F. Respirations quick, slight cough, and expectoration streaked with blood. A few râles in lungs.

It was evident he was suffering from suppurative lepto-meningitis and sinus thrombosis, possibly with infective inflammation of lung.

Operation (6th Jan., 1892).—The mastoid antrum was exposed and found filled with granulation tissue and fœtid pus. The tegmen was eroded and granulation tissue protruded through it from the dura mater. On removing the tegmen a quantity of pus—about three drachms—escaped from between the dura and the skull. The dura was covered with granulation tissue, and after its removal a small softened area was seen, through which the probe entered. The inner membranes were soldered to this patch.

They were opened, and the surface of the brain was seen to be in an inflamed condition, red and injected. There was, however, no pus inside the brain.

The sigmoid groove was opened, a thin shell existing between the enlarged antrum and the groove. It was covered with a layer of plastic lymph. The sinus was patent, though its walls were thickened.

The pus having been removed, the cavity washed out and rendered aseptic, the whole aperture was filled with boracic acid and iodoform, and the wound dressed. The temperature became normal at the end of forty-eight hours, and the wound healed slowly by granulation tissue. Patient made an uninterrupted recovery.

Cases VIII.–IX. *Summary of two cases of extradural abscess from necrosis of tegmen or sigmoid groove originating in otitis media.*

The following case illustrates direct inflammatory extension of infective disease from the middle ear to the cerebellar fossa, through the medium of the bone. The patient was five years of age, and had suffered for over four months from otitis media, which had commenced after an attack of measles. The tissues over the mastoid had been œdematous, and had been incised

before he came into the hospital. When admitted not only fœtid otorrhœa, but also symptoms of cerebro-spinal meningitis existed, including high temperature, retraction of the head and neck, rigidity of the arms, intolerance of light, extreme fretfulness, while he resented any interference. The antrum of the mastoid was opened, and, the contained fœtid pus having been washed out, a portion of the internal table of the skull forming part of the sigmoid groove was found to be necrosed and detached from the surrounding bone, but was held in position by granulation tissue. When this portion of bone was removed two drachms of pus escaped from between the sigmoid sinus and the skull. The sinus, which was somewhat separated from the bone, was covered by a layer of yellow plastic exudation to which fluid pus was adherent. The portion of necrosed bone measured a quarter of an inch in both diameters, and was in direct contact with the diseased antrum on one side and with the sinus on the other. After operation the wound healed by granulation tissue, and the patient made a rapid recovery.

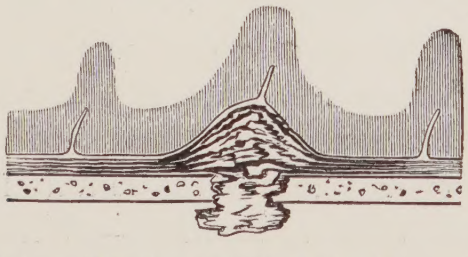
In another patient, a girl aged nineteen years, the same condition was found (as far as the loose necrosed portion of bone was concerned) in the tegmen tympani, on the removal of which about three drachms of pus escaped. She had symptoms of localized meningitis, which disappeared soon after the removal of the pus.

These are typical of ten cases operated on in the author's practice, in which detached bone existed between the middle ear and the cerebral or cerebellar fossa, all originating in *otitis media purulenta*. There were others of gross necrosis, where the greater part or whole of the mastoid was necrosed along with the internal ear.

Had those cases been left to proceed in their usual course purulent meningitis, thrombosis of the sinus, or abscess of the brain, would in all probability have resulted.

The mode of infective involvement of the membranes and the brain.—Very frequently, if not always, there is a thickening of

FIG. 29.—SCHEMA OF INFECTIVE INVASION OF MEMBRANES.



Thickening of dura by cellular infiltration projecting on its visceral surface and indenting the brain. Granulation mass projecting from its external surface through osseous erosion.

the dura mater at the point from which the granulation tissue springs. It is hypervascular, œdematous, occasionally filled with new cells, and softened.

Involvement of the middle cerebral fossa.—Where the tegmen

tympani (see Fig. 30) is involved, especially in severe cases, the dura has on its visceral side, at a point corresponding to the osseous erosion, a raised projection of granulation cells and plastic effusion, generally somewhat conical in shape, with its obtuse extremity pointing upwards (see Fig. 29), and forming an indentation on the surface of the brain, the pia mater being usually adherent to it.

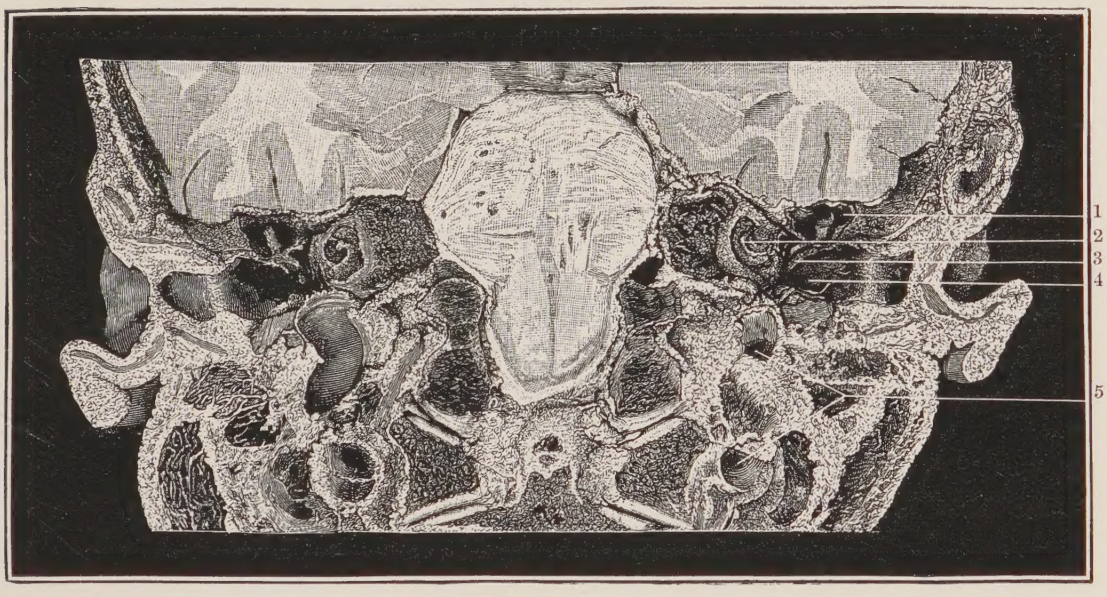
In this way the veins and lymphatics of the pia become directly affected by the intimate contact of the pia mater with and its adhesion to the inflammatory exudation mass projecting inwards from the dura. In some instances the branches of the cerebral arteries supplying the temporal lobe have become thrombosed, and as they are terminal vessels, with little or no anastomosis, minute emboli from this peripheral nidus are deposited in the interior of the white cerebral substance, round which pathogenic processes extend, giving rise to cerebral abscess. The infective processes may also extend by way of the perivascular sheaths from the periphery into the brain. This opinion is supported by the fact that cerebral abscess is far more frequently found in the white substance than in the cortex, though abscess in the white cerebral substance may also be formed by venous thrombic extension from the periphery inwards against the stream.

With the exception of the extension of the granulation masses through the tegmen, all the other phenomena just described may occur without an actual visible aperture in the tegmen itself, though the bone may be eroded, discoloured, and occasionally necrosed. When the bone is still living some of its vessels are thrombosed and convey the pathogenic organisms from the middle ear to the thickened dura. Occasionally, especially in children, the disease from the middle ear extends to the dura through the petro-squamosal or other suture, many of the vessels being thrombosed and the dura adherent at that point. Close to the petro-squamosal suture osseous erosion is sometimes seen, which behaves just as the erosion nearer the middle line does.

How the cerebellar fossa is attacked.—When the seat of the osseous erosion is the sigmoid groove (see Figs. 32, 34, and 35), the external wall of the sinus throws out its granulation tissue in a similar manner to that of the dura over the tegmen tympani. In some instances the sigmoid groove is separated from the mastoid cells by a plate of bone even thinner than the tegmen tympani.[1]

[1] See Atlas of Head Sections, Plates 18 and 19.

FIG. 30.—THE RELATIONS OF THE BASE OF THE TEMPORO-SPHENOIDAL LOBE TO THE TYMPANIC ATTIC.



1. Attic of tympanum with ossicle. 2. Cochlea. 3. Membrana tympani. 4. External auditory meatus. 5. Carotid artery.

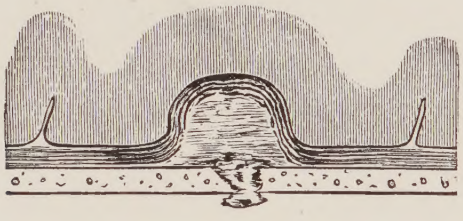
Coronal head section, male, 5 years, viewed from before, passing through the external and middle ear, with the attic of the tympanum, the cochlea, the bases of the temporo-sphenoidal lobes, the pons and medulla.

The thin wafer of bone forming the tegmen tympani and antri is the only barrier between infective processes generated in the tympanum and the brain membranes. The ascending sulcus whose base rests upon the dura covering the tympanic attic and antrum is the one into which infective processes most frequently spread, and the adjacent white brain matter is the initial area of abscess in the temporo-sphenoidal lobe.

To face page 72.

An extradural abscess may form between the sinus and the bone, and it may project on either side, in front and internally, along the posterior aspect of the petrous bone, or behind the sinus toward the occipital bone. The inner aspect of the sinus participates in the inflammatory process, which has given rise to the granulation tissue projecting into the mastoid cells. It can scarcely fail to do so, as the layer of the dura dividing the blood-current from the bone is only half the thickness of that covering the tegmen, where a similar process ensues. The result of the internal surface of the outer layer becoming inflamed, is the formation of a thrombus in the sigmoid sinus, the slowness of the blood-current at this spot greatly favouring the thrombic formation. The inflammation of the walls of the sinus not only

FIG. 31.—SCHEMA OF EXTRADURAL ABSCESS.



Bulging of softened dura toward visceral side of extradural abscess and osseous erosion, with granulation tissue springing from bone.

extending longitudinally but also circumferentially, the side nearest the cerebellum becomes implicated. The thickening of the coats of the vessel, and the thrombosis of a part of its lumen, may remain for a long time stationary before further mischief ensues. The irritation maintained by successive pathogenic attacks upon the outer wall is apt to cause softening, destruction of the original wall of the sinus, and of part of the newly formed granulation tissue, so that there may be a minute perforation, occasionally a very wide one, in the outer wall of the sinus, through which the disintegrating contents of the vessel may find an entrance into the mastoid cells. As a rule no blood escapes, as the sigmoid sinus is in such cases generally doubly occluded, first by one portion of adherent thrombus above, near the lateral sinus, and again by another below toward its terminal portion, before it joins the jugular bulb in the jugular fossa. The centre of the thrombus corresponding to the perforation through the mastoid cells is that which is most liable to disintegration. When the whole circumference of the sigmoid sinus is involved, and is the seat of saprophytic and pathogenic organisms, the cerebellar veins

become affected, and so convey by thrombic extension pathogenic organisms to the cerebellum. By a process of thickening of the dura on the side of the cerebellum similar to that described as having occurred in the dura over the tegmen, the cerebellar arteries and their perivascular sheaths are also liable to be involved, and to transmit emboli into their terminal portions in the interior of the cerebellum.

A cerebellar abscess may form by direct extension through the dura on either side of the sigmoid sinus, the dura mater there passing through the same processes as have been already described. The majority of cerebellar abscesses are in direct contact with the sigmoid sinus. Inflammation may in exceptional cases travel backward from the middle ear through the internal ear to the cerebellum along the nerve sheath of the facial and the auditory nerves, but when it does travel by this path it is much more apt to give rise to lepto-meningitis than to cerebellar abscess. Again, when the *aqueductus Fallopii* is exposed in the middle ear, pathogenic organisms may pass along the nerve sheaths of the facial, either toward the cerebellum, or upward and forward through the foramen enclosing the great petrosal nerve. Here, again, the inflammation extending along this path gives rise to lepto-meningitis rather than to abscess of the brain.

If the pathogenic matter enters the subdural space before adhesions with the pia mater have been formed, lepto-meningitis is apt to be set up, in which case the whole of the cerebral and cerebellar meninges are apt to be involved, the inflammatory action spreading with the greatest rapidity along the areolar tissue of the arachnoid. The adhesion of the inner membranes to the dura at these points of irritation, even though abscess in the brain ensues, is, therefore, a conservative action preventing the graver evil, general infective lepto-meningitis.

Reasons for caution in removal of granulation tissue from the middle ear.—It has been mentioned that the removal of these masses of granulation tissue—"polypi"—from the middle ear has been followed by meningitis or abscess of the brain. The probable explanation of this is, that as long as the granulation tissue remains intact, it forms a natural barrier to the absorption of pathogenic products. By removing this granulation tissue in the midst of infective matter, the fresh wound made becomes affected, and so the pathogenic organisms gain an entrance into the intracranial tissues. The same thing might no doubt also

FIG. 32.—EROSION FROM INFECTIVE PROCESSES IN KNEE OF SIGMOID GROOVE.

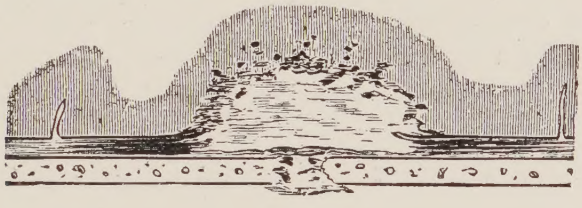
Infective erosion in knee of sigmoid groove—the part of groove most commonly involved in extension of infective processes from the mastoid antrum and cells. The darkened bone is partly hidden by the overhanging ledge of the petrous bone.

To face page 74.

result were fresh pathogenic organisms introduced from without by unclean instruments or otherwise. When the granulation tissue actually sprouts from the dura through the bone, and this granulation is abrupted, no doubt the infective matter then entering the open mouths of the exposed vessels would have a direct passage into those of the pia, through the plastic inflammation of the dura.

Subdural abscess.—Ulceration of the brain.—An abscess may form, however, between the pia mater and the dura, being circumscribed by the adhesion formed between these two membranes. In such cases there is superficial inflammation of

FIG. 33.—SCHEMA OF ULCERATION OF BRAIN.



The membranes are disintegrated, and a tortuous channel exists between them and the exterior of the skull.

the brain, and not infrequently this results in brain ulceration, the molecular disintegrating products of which accumulate between the brain and the dura. Under these circumstances the plastic exudation formed on the inner side of the dura softens and degenerates; the membranes participating in the softening process may become eroded, and even perforated, so that pus may escape on the external surface of the dura, or, in the case of an osseous perforation, through the tegmen tympani.

CASE X. *Ulceration of brain, with abscess between surface of brain and skull —arising from chronic otitis media. Opening of base of skull, and evacuation of abscess. Peculiar position of mastoid antrum relatively to the base of skull. Recovery. Coming under observation three weeks after beginning of attack. Duration of illness six weeks.*

E. M'C., aged 19 years, miner, was admitted into Royal Infirmary on 1st June, 1891, suffering from superficial ulceration of the brain connected with chronic otitis media.

History.—*Heredity.*—He was one of a family of thirteen, parents were alive and healthy; three of his brothers died in childhood, one from smallpox, one from "wasting of the bowels," and a third from croup. One of his brothers had "a running ear."

Personal.—Patient has been a delicate boy, had not walked till about three years old, subsequently he improved a little, though continuing weak. At age of fourteen, he commenced work, which he had been able to continue till the last few weeks.

Eighteen months ago he had a discharge from the ear following a sore throat. This sore throat had troubled him occasionally from the time he had measles when five years of age. A year after the first appearance of the otorrhœa, he again was troubled with sore throat, followed by renewal of the discharge from the ear which has persisted since. At first it was copious and watery, but recently it had become purulent and foul-smelling.

Present Illness.—Three weeks ago he was affected with a very severe otalgia and headache on left side, and had a slight shivering which compelled him to stop work for a week. The headache subsiding, he resumed work for six days, at the end of which time it recurred, and he had to desist.

The day before admission he had a severe headache on the left side which was followed by a series of fits (six in number). In these the mother stated that the head and eyes were turned to the right; nothing beyond general convulsive movements was observed in the limbs by the friends. All those fits occurred within six hours, during which he was unconscious. There was no history of vomiting nor of giddiness, though, as to the latter, it is doubtful whether the patient clearly understood the meaning of the term. His bowels have always been constipated.

Condition on Admission.—Patient lay still when undisturbed, could answer questions quite intelligently, though there was a general obscuration of his brain functions. There was no rigidity nor paralysis of the limbs, though the muscular movements were feeble. The reflexes were normal.

Sight.—Sight unaffected. He had an intolerance of light, and preferred to keep his eyelids closed. Slight congestion of both discs. No ocular paralysis, both pupils sluggish, left slightly dilated.

Hearing.—Right side normal; aerial conduction absent on left side, but bone conduction normal. Right membrana tympani normal. Left membrane wanting, and the tympanic cavity was filled with granulation tissue and foul-smelling pus.

There was a mitral obstruction murmur present, and the urine showed a cloud of albumen. Pulse was regular, 62 per minute, very soft. Respiration, 15 per minute. Temperature, 98° F. He had pain over the left mastoid and along the course of the jugular.

Operation (1st June, 1891).—The mastoid antrum was exposed. On this being done, the same formation of parts was found as is described in Case XXVI. The antrum was considerably enlarged and filled with granulations and pus. The tegmen tympani was eroded, and pus issued from the erosions. The base of the skull (including the tegmen tympani) over the lower and outer temporo-sphenoidal convolution was laid bare. The dura mater was found to be softened and ulcerated over an area of about half an inch. The brain tissue, which was bathed in pus, was also in a state of superficial ulceration. The area involved was about $\frac{1}{4}$ inch

FIG. 34.—PATHOLOGICAL EROSION BETWEEN CEREBELLAR FOSSA AND EXTERIOR OF MASTOID.

The external orifice of an osseous erosion about line of masto-squamosal suture which forms a passage between the cerebellar fossa and the exterior of skull through which pus from a cerebellar abscess escaped. The osseous sinus passes downwards and backwards into sigmoid groove. The posterior extremity of the osseous erosion corresponds to the knee of sigmoid groove—which is about a quarter of an inch behind border of external osseous meatus.

To face page 76.

in diameter. Three drachms of pus were removed. A trephine disc was elevated from the squamous bone near the base of skull to permit the inspection of the condition of the dura above. That membrane was found healthy, and the inflammation not having extended so far, the disc of bone was returned. The sigmoid sinus was also laid bare, as the bone in its vicinity was dark-coloured. There was no actual pus found between the sinus and the bone, but the dura was there thickened and in part covered by plastic exudation. The parts were dressed in the usual way.

Subsequent Progress.—From the time of operation patient's symptoms improved; he became much brighter. Temperature, pulse, and respiration became about normal, and, with the exception of the left pupil which continued slightly dilated and somewhat fixed for about three weeks, he was in a healthy condition.

Three weeks after operation, he was dressed for the first time, when the bone surrounding the large cavity was found to be covered with granulation tissue. Subsequently he made an uninterrupted recovery.

CASE XI. *Ulceration of the brain, superficial cerebral abscess formed between meninges and temporo-sphenoidal lobe, arising from tubercular processes in the mastoid and petrous bone and accompanied by tubercular lepto-meningitis and enteritis. Duration of disease unknown. Died from enteritis forty-seven days after operation. The following is an abstract of case.*

M. M'I. (female), aged 9½ years, admitted into Children's Hospital, Glasgow, in November, 1887, affected with lepto-meningitis of tubercular type, with extensive tubercular destruction of mastoid and petrous bone; also ablation of middle ear and ulceration of base of temporo-sphenoidal lobe.

History.—The hereditary and personal history was very meagre.

Condition on Admission and Operation.—The patient was extremely emaciated, and suffered from a characteristic attack of chronic meningitis of a rather diffuse kind, accompanied by a profuse infective discharge from the right ear, as well as from a sinus in the mastoid which led into a large hiatus in the petrous and mastoid bones, surrounded by caseating debris and crumbling bone. The middle ear as such had disappeared. The tegmen tympani was ablated, and the dura converted into fungating granulations projected from the roof. The sigmoid sinus was likewise exposed. These points were seen after the external mastoid shell had been removed and the contents of the cavity had been cleared. After repeated cleansing of this cavity and attempting to render it aseptic, more pus exuded from it than was likely to issue from the surrounding granulation tissue, and on several occasions the usual quantity was somewhat suddenly augmented. An exploration was therefore made in the granulation tissue projecting from the dura covering the roof of the cavity, when an aperture was found which easily admitted the probe through the dura into a cavity formed by the meninges and the base of the brain. A portion of bone was removed from the base of the squamous and the third

temporo-sphenoidal gyrus was exposed. The brain was here seen to be in a state of ulceration, the pus from which was retained by the soldering of the membranes at the periphery of the ulcer. This small abscess cavity communicated with the outside of the skull by an aperture in the dural granulation tissue which covered the gap in the tegmen tympani. The abscess cavity was nearly empty, free exit for the pus being obtained through the sinus.

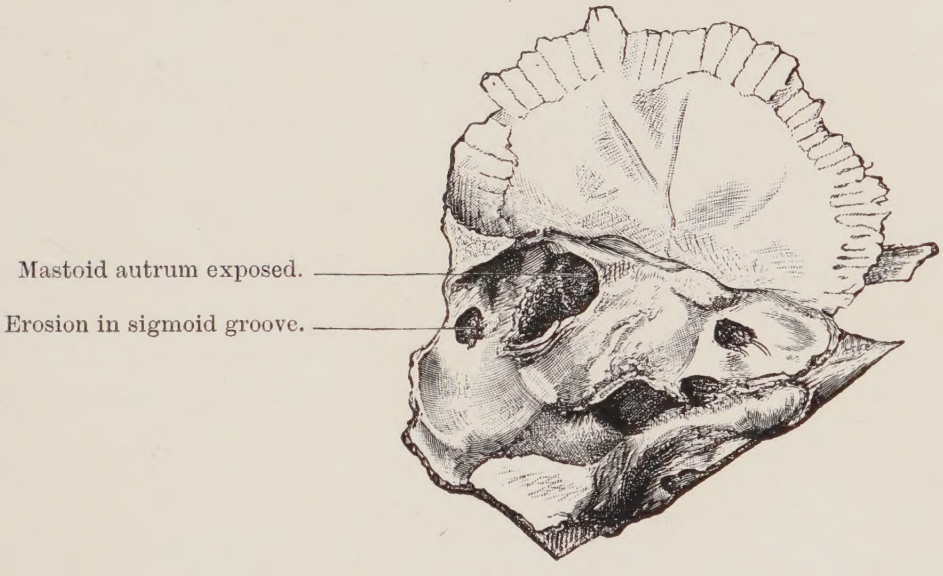
This sinus was dilated to prevent the possibility of re-accumulation of pus. Subsequently the whole cavity filled with granulation tissue which was in process of cicatrization. The pus secretion greatly diminished, and was never greater than what could be produced by the granulation tissue itself. The meningitis however persisted, though for a time there was a lull in the symptoms. Shortly afterwards an attack of what ultimately proved to be tubercular enteritis came on and rapidly reduced her remaining strength. She died on 27th December, 1887, forty-seven days after operation.

Post-Mortem Examination.—*Brain.*—There was marked lepto-meningitis all over the brain, miliary tubercle being scattered over the pia, especially abundant in the left Sylvian fissure, several of the lobes being soldered. Between many of the convolutions there was an excessive accumulation of slightly turbid serous fluid, giving a milky colour, especially noticeable at each side of the veins. The left lateral ventricle was greatly distended with turbid serum, the right though not so distended also contained an excess of fluid. The brain substance generally was firm and healthy. The base of the temporo-sphenoidal lobe was at one point adherent to the granulation tissue which occupied the gap in the tegmen tympani. Traction on the granulation tissue showed the firm adhesion had to the third temporal convolution. On section through granulation tissue and brain it was seen that connective tissue formations penetrated some distance into the brain, where they were met by a puckering in the brain tissue ; in the midst of the latter there was a small granulation nodule containing a drop of tenacious glairy yellowish fluid. This was all that remained of the superficial abscess at the seat of the brain ulceration. The osseous hiatus was very great, involving the whole of the mastoid cells, a large portion of the petrous, including the whole of the tegmen,—a very small part of the osseous portion of the Eustachian tube remaining,—and exposing by large apertures the sigmoid sinus and the jugular fossa.

Cases XII., XIII. *Summary of two cases of ulceration of brain with superficial brain abscess.*

The two following cases are instances of extensive destruction of the petrous bone, with ulceration of brain, and abscess formation between the membranes and the brain surface. The dura covering the tegmen tympani was softened, flocculent, eroded and perforated. Through these perforations pus exuded from the subdural cavity. On the dura being freely opened a few drachms of pus escaped. The brain surface exposed was ulcerated. It is unusual to find cerebral abscesses, even small superficial ones, such as these

FIG. 35.—INFECTIVE EROSIONS IN KNEE OF SIGMOID SINUS.



Left temporal bone viewed from within and behind, showing two erosions, a large one at knee of sigmoid groove communicating with mastoid antrum, and a smaller one in sigmoid groove communicating with exterior of skull, through either of which the pus from the cerebellar abscess escaped.

To face page 78.

were, in connection with very great destruction of bone in young children. The dura is often exposed in them, but it is covered by a mass of granulation tissue which protrudes into the tympanum, and so far acts as a protecting medium.

M. B., aged 22 months, admitted to Royal Infirmary, 24th June, 1887, suffering from symptoms of cerebral disturbance, paralysis of the seventh nerve, and pus issuing from the right ear.

The child had had slight suppuration in the right ear for a considerable number of months, and even prior to that used to cry frequently from pain, although the mother could not state definitely whether the pain was in the ear or not. During the last four months the discharge had a very offensive odour, and had been very profuse. Within the last month the pain became greatly aggravated, the child screeching constantly when awake, and often roused by it when asleep. The paralysis of the seventh nerve had been observed for six weeks prior to admission. The question as to whether cerebral abscess was present was raised, but was difficult to answer owing to the age of the patient. The child was greatly emaciated, constantly crying (which exhibited the paralysis of the seventh), had high temperature (103° F.), and was altogether in a precarious condition. There was a profuse discharge from the ear, and on examination the mastoid and the petrous portion of the temporal were both found to be necrosed.

Operation (29th June, 1887).—An incision was made at the back of the ear over the mastoid. The latter was found necrosed and loose and was removed ; then a considerable portion of the petrous bone was extracted, having undergone similar destruction. The dura mater thus exposed was thickened, flocculent, and covered by a layer of pus. When this was removed, a small quantity of pus was seen to ooze from its interior. A probe introduced by that aperture was found to enter a cavity in the temporo-sphenoidal lobe. The aperture in the dura mater was enlarged, and gave vent to two drachms of pus. The surface of the brain was eroded, softened, and in a state of ulceration. The cerebral abscess cavity was afterwards washed out with boracic solution, and a drainage tube inserted. A wood-wool dressing was applied outside.

The temperature on the evening after the operation was 103·2° F., but next night it had fallen to 100·2° F., and afterwards remained normal throughout. The drainage tube was removed on the sixth day, when the cavity was seen to be covered with granulation tissue. The wound was closed at the end of a month.

27th November, 1887. Seen to-day, and found well.

J. B., aged 2 years, admitted into Royal Infirmary, 29th August, 1887, suffering from profuse, foul-smelling, purulent discharge from the left ear of four months' standing, facial paralysis, and a general condition of marasmus, owing no doubt to the excessive pus secretion, the amount of which coupled with other symptoms suggested cerebral abscess, secondary to the condition of the tympanum. On examining the part through the external ear, extensive necrosis of tegmen was discovered.

Operation (30th Aug., 1887).—An incision was made over the mastoid, exposing a necrosed mass which was comparatively easily removed. After its removal a second portion of the petrous bone, in front nearer the middle line and involving the tympanum, also came away very easily.

During the removal of these pieces of bone, vent was given to a considerable quantity of pus,—several drachms. The dura mater was seen to be flocculent, and an eroded aperture was found in it which led into an abscess cavity in the temporo-sphenoidal lobe. This cavity was washed out with boracic solution, and as the aperture in the dura mater was sufficiently large to permit free drainage, no tube was introduced. The surface of the brain was shaggy, softened, and evidently ulcerated.

Highest rectal temperature after operation: 99·6° once only, 101° F. ten days after operation.

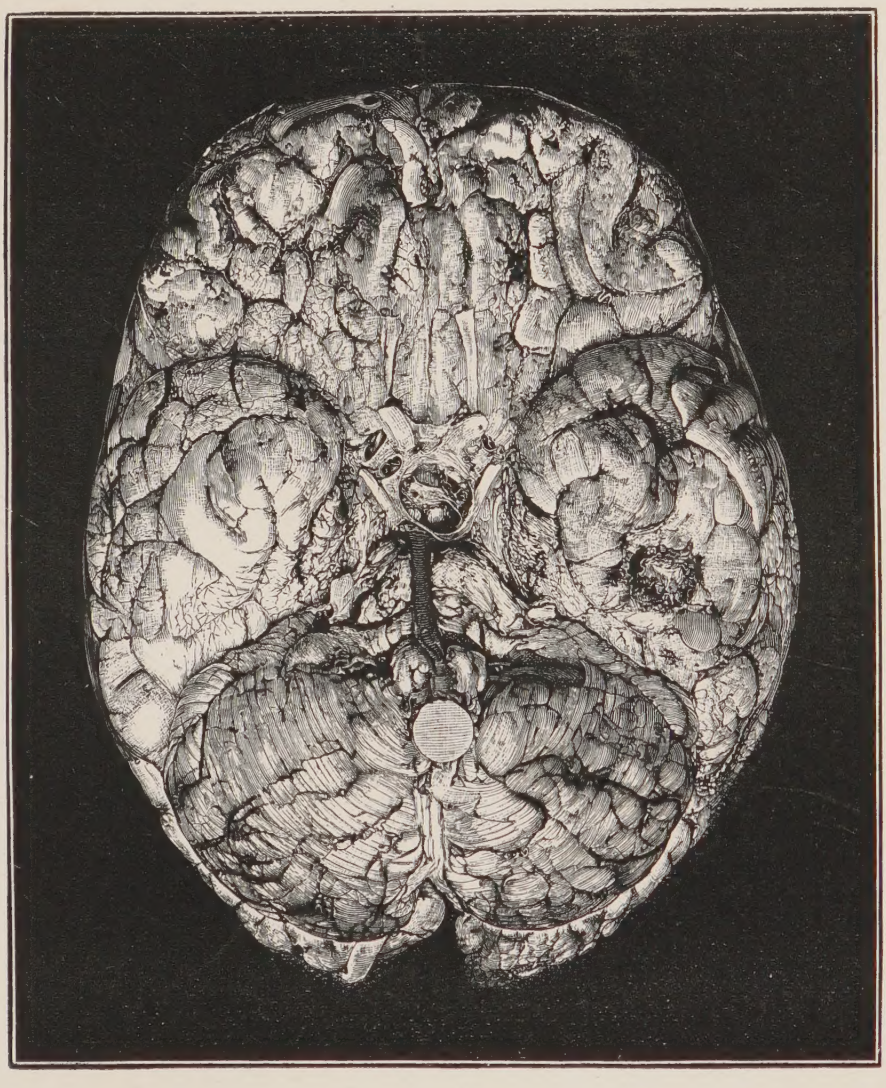
29th November, 1887. Seen to-day, and found well.

Both of these cases were seen on several occasions afterwards, one at intervals during two subsequent years, the other during five years. On each occasion they were found perfectly well as far as the head and ear were concerned. One of them (the former) had subsequently tubercular manifestations in both ankles and in several of the fingers. These parts were operated on, and patient recovered.

The formation of abscess in the middle cerebral fossa.—It has been seen that the infective disintegration originating in the tympanic cavity does not affect its walls equally, but extends principally in two directions, upwards and backwards. The anatomical arrangement of the roof of the middle ear, as may be seen in section (Fig. 30), affords but a slender barrier against infective invasion. Though any portion of the tegmen may be involved, the tegmen antri and tympani are the parts generally affected in adult life—seldom the tegmen Eustachii; while in children, though the infective matter frequently passes through erosions in the tegmen, yet it often does so by the petro-squamosal suture. In the majority of instances the infective path, when carefully looked for, is marked by a lesion visible to the naked eye, and often by a papulated eminence in the membranes (Fig. 29), which leaves an indentation in the brain, in the periphery of which the membranes are soldered, while at the point of contact of this conical projection with the brain, the cerebral tissue is inflamed and softened, as is shown in Fig. 36.

Frequently the infective processes extend along the pial vessels which dip into the sulcus, the base of which rests over the tegmen (see Fig. 30), in which case the cells forming the infective nidus are often accumulated toward the central extremity of the sulcus, and enclosed within it. In removing the brain at post-mortem

FIG. 36.—COMMENCING INFECTIVE INVASION OF LEFT TEMPORO-SPHENOIDAL LOBE.



Indentation of brain corresponding to conical-shaped projection of infiltrated membranes, arising from infective processes extending through the tegmen tympani. The brain at this spot is softened and inflamed—the encephalitis extending for about quarter of an inch into the brain substance. There is as yet no abscess.

Photographed from fresh brain—patient having died from infective sigmoid sinus thrombosis.

To face page 80.

examination, unless this sulcus is opened the infective deposit within it is overlooked.

In either case, the softened brain tissue, in contact with the infective nidus, is prone to purulent disintegration forming a cerebral ulcer, and the pus ensuing from the ulceration being confined between the soldered membranes and the brain will probably be described as an abscess, especially when met with during an operation. The formation of a true cerebral abscess, however, occurs by a further extension from this pial accumulation and softened cerebral tissue along the paths of the cerebral vessels, which penetrate the white substance of the brain. The infective virus acting upon the vessels, and causing œdema of the brain with leucocytal exudation, often mingled with extravasation of red blood corpuscles, soon forms an area of purulent encephalitis. In the majority of fully-formed abscesses in the temporo-sphenoidal lobe, an opening into the abscess cavity exists at the base of the brain, which indicates the point of brain indentation caused by the infected conical projection in the membranes (see Figs. 37, 38, 39, 40, 41, 42). These openings may be regarded as the area of initial invasion, and not merely as due to a secondary process of absorption of the abscess wall. Such openings in the base of the brain connected with abscess in the temporo-sphenoidal lobe are almost always located in the same place as shown in Figs. 37 and 38, so that, as a rule, in cases of cerebral abscess the path by which the infective processes spread from the tegmen to the brain can be traced, and the majority of abscesses develop by direct extension from the infective seat. It is therefore of great importance, in the treatment of such cases, besides dealing with the abscess, that the point of origin of the infective processes in the tissues should be sought for and their paths of extension traced, in order that they may be eradicated.

The statement that disease of the tympanum causes cerebral abscess chiefly, while disease of the mastoid cells causes cerebellar abscess, is misleading, inasmuch as the tympanum is diseased in both forms; and in the majority of cases in which the mastoid antrum and cells are affected, grave intracranial disease of the middle fossa results. The tegmen tympani is often eroded at the same time as the involvement of the sigmoid sinus occurs, and cerebral abscess in the temporal lobe is not infrequently met with, along with infective sigmoid sinus thrombosis.

Why intracranial affections due to otitis media may be more

frequent on the right side than on the left.—It has been stated that the right side of the head is more often affected by intracranial sequelæ from otitis media than the left. If so, this is probably due to the greater size of the lateral and sigmoid sinus on the right side, as compared with its fellow on the left. Consequently, the right sigmoid sinus encroaches more upon the petrous and the mastoid portions of the temporal, especially at the sigmoid knee, and the distance between the lower border of the tympanum and the antrum on the one hand, and the sigmoid sinus on the other, is less than between the corresponding parts on the left side. This is especially noted in brachycephalic skulls. The initial disease —otitis media—shows no preference for one side more than the other. Though there is a preponderance of right over left intracranial sequelæ, the difference is not one which is very marked when the range of observation is sufficiently large.

Cerebellar and cerebral abscesses may empty themselves through osseous erosions.—When the internal wall of the mastoid cells has been eroded so as to expose the sigmoid groove, extradural pus escapes into the cells, and would do so freely were it not for the partially occluding granulation tissue. When, in addition to the osseous aperture, there is a softened and eroded dura, pus from the subdural space,—or, when that space has been obliterated, from the cerebellar abscess itself,—may gain an exit by way of the mastoid cells to the middle ear, and so escape by the already perforated tympanum. Erosions may take place in the external wall of the skull from the sigmoid groove or its vicinity, when the pus will pass directly into the external soft tissue and may escape through a sinus in them. This is frequently seen in children. Perforations of the tegmen tympani act in a similar way, as channels of escape, for extradural pus, and when the dura has been eroded, for the contents of the cerebral abscess. These points have been verified by the observations of the author, first, in patients, in some of whom the cerebral, and in others the cerebellar fossæ, being the seats of disease, were exposed, when the osseous erosion, the aperture through the softened dura, and the escape of pus directly from the abscess in the brain were observed during operative measures undertaken for the relief of the disease. Second, in one instance, a child suffered from profuse otorrhœa of a very chronic kind, though he was able to go about. At a dispensary he had an injection of nitrate of silver forced into his middle ear, in hope

of curing the otitis media. As an immediate consequence, the child became seriously ill with brain symptoms, when it was found that the tegmen had been perforated and a cavity in the temporal lobe exposed, into which the nitrate of silver injection had been forced. The child died from acute encephalitis, set up in the periphery of this cavity. Third, by post-mortem examination of parts sent to author, and of some occurring in his own practice. Fourth, by the detailed accounts given to the author of a patient who had suffered from childhood from chronic otitis media, and who became affected with an illness closely resembling that of cerebral abscess, passing on to unconsciousness lasting several days, when she was considered to be at death's door. It was then discovered by the somewhat sudden increase of the foul smell, that pus was "pouring" from the affected ear, and had already saturated a pillow before it was observed. The patient regained consciousness shortly thereafter, and slowly recovered her intelligence and her strength. No physical examination of the parts were ever made in this case further than by the auriscope, which showed that the membrana tympani was ruptured and granulation tissue projected from the middle ear. The amount of pus which somewhat suddenly escaped, and the mental symptoms exhibited, could hardly be accounted for by an extradural abscess over the tegmen. This lady, when seen by me, twenty years after the described attack, was in good health, though deaf in the affected ear, which still discharged a little pus. She was very intelligent and active, though, if suddenly or abruptly addressed, she said she required some moments to collect her thoughts, and at such times seemed to others "dazed." She complained of pain in the head and dizziness when she stooped and rose up suddenly. Since her early severe illness she had been several times (at least twice) in bed for weeks with what she called 'cold,' at the beginning of which she had a shivering, the otorrhœa ceased, pain in the same ear and side of the head ensued, and she was semi-delirious for days. All these symptoms disappeared on the escape of a 'free flow' of pus from the ear.

It must be admitted that there are cases in which abscess of the brain may find an exit through the erosions set up by the ulceration. It is very rare that they are cured in this way, as even in the case quoted above, the evidence points to the probable existence of a small sized encysted abscess in the brain, which had a tortuous communication with the middle ear. As a rule these

abscesses cannot empty themselves owing to the tortuous passages through the erosions filled with granulation masses, and on account of the resistance in their walls when encysted. Most of them end fatally when not relieved surgically.

Addenda to mastoid pathology.—In young infants, tubercular disease in the middle ear is apt to pass through the unossified portions of the temporal and affect the membranes with tubercular meningitis. (There is one such case quoted under the heading of tubercle in middle ear.) In those cases tubercular glands, behind the ear superficially (submastoid) and also deeper ones, involving the sheaths of the great vessels of the neck, are frequently found.

In young children, infective disease of the tympanum and the antrum is prone to induce suppurative lepto-meningitis, generally basal, and especially involving the cerebellum and basal gangliæ. The soft membranes become affected through the numerous veins and nerve sheaths. This has been found by the author in cases where there was little secretion from the ear, but where the post-mortem showed pathogenic destruction of the middle ear and antrum, and where the labyrinth was invaded by the inflammation. In infants the aqueductus Fallopii is much more exposed to infective action than in adults, owing to its imperfect ossification. Thus facial paralysis occurs much more easily in infants, and pathogenic processes are more prone to spread from the middle ear along the nerve sheath to the base of brain.

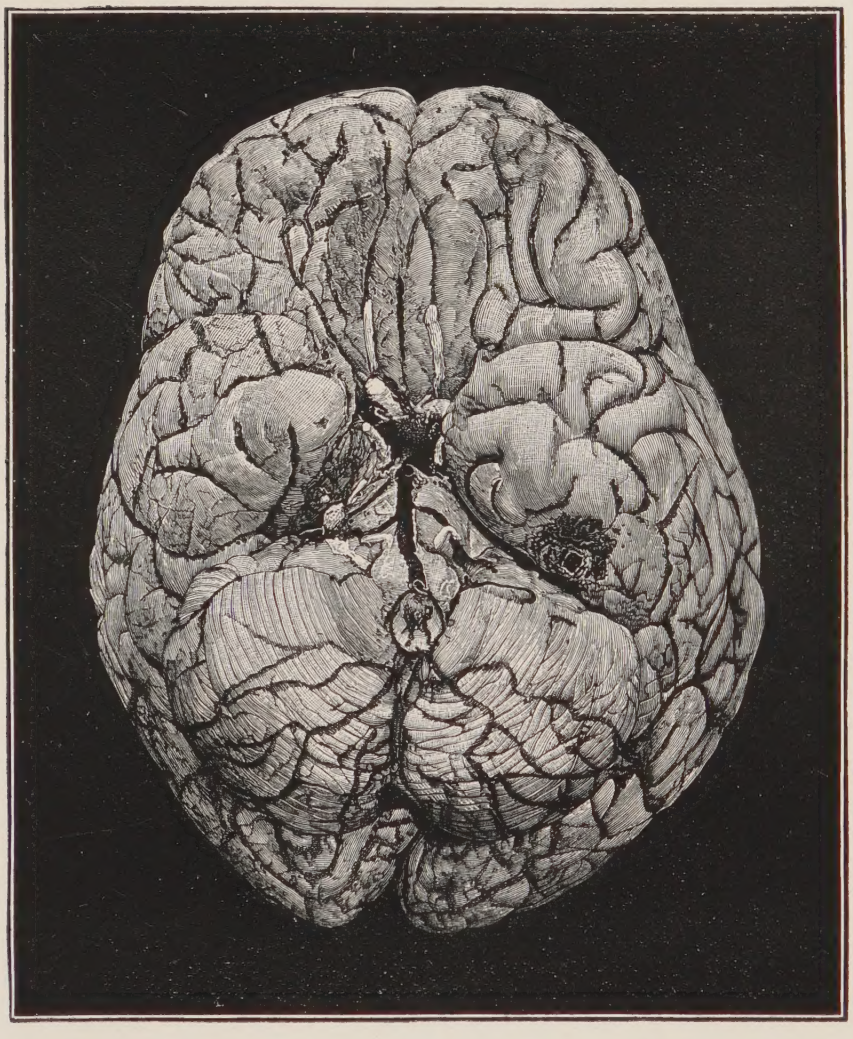
In children who have had acute periosteal attacks, and in some who have suffered from tubercle or hereditary syphilis, large necrosed masses occur in the mastoid. Often the greater portion of the mastoid with the exception of its tip, and occasionally portions of the petrous and squamous bones, have been shed in this way.

Purulent inflammation in the tympanic cavity may extend into the Eustachian tube, in the walls of which an abscess may form and ultimately produce gravitation abscess in the naso-pharynx. These retro-pharyngeal abscesses are apt to occasion respiratory difficulties and impede deglutition. The pus accumulating about the articulation of the lower jaw may, in rare cases, ultimately cause erosion of the capsule.[1]

Acute lepto-meningitis.—When acute inflammation is once established in the loose arachnoid meshwork, it spreads rapidly

[1] Pus in mastoid cells may escape by erosion into Digastric Groove and form abscess under deep cervical fascia. See Atlas, Plates 8 and 18.

FIG. 37.—ABSCESS IN LEFT TEMPORO-SPHENOIDAL LOBE.



Aperture in base of brain, connected with abscess in temporo-sphenoidal lobe, and with granulation mass springing from the membranes and causing an indentation on cerebral surface. The tegmen antri and tympani were eroded.

From photograph.

Follows page 80.

and widely, until the whole cerebro-spinal subdural and intraventricular spaces are involved, as well as the pia mater, and, in most instances, the superficial layers of the brain. The sheaths of the cranial and spinal nerves are likewise implicated for a considerable distance outside the central cavities. The brain and the spinal cord float in the cerebro-spinal fluid, in the same way as a specimen would do in a bottle specially moulded to fit the form of the contained object. This fluid is nearly continuous throughout. When pathogenic micro-organisms are inoculated into that fluid they spread with remarkable rapidity, finding therein a suitable temperature and abundant pabulum for their development. Within twenty-four hours from the first rigor, symptoms of involvement of the whole cerebro-spinal system may be manifested, and in virulent forms death may supervene within a few days. While inflammations confined to the brain itself are serious, they are much more limited and, even in acute forms, slower in their course than lepto-meningitis. The extent of the surface involved, and the derangement of the function of the cerebral cortex produced in lepto-meningitis, renders it one of the most formidable of all brain affections.

Acute serous lepto-meningitis.—Acute lepto-meningitis may be of either a serous or a purulent type. When simply serous, the hyperæmia is followed by an exudation of clear serum often containing flakes of fibrin and occasionally a few leucocytes, and possibly even a few pus corpuscles. The leucocytes are found in the perivascular sheaths of the cortical bloodvessels. The serous fluid may accumulate so quickly as to produce pressure upon the cerebral bloodvessels, ending in their occlusion and the rapid death of the patient. When the serous lepto-meningitis persists, the ventricles become greatly distended—thus forming internal hydrocephalus—and press the brain against the dura, flattening the convolutions. Occasionally, such meningeal effusion of serum forms an acute meningeal or external hydrocephalus. Instead of the acute serous lepto-meningitis extending all over the subdural space it may be localized. This especially occurs when inflammation extending from without involves the visceral aspect of the dura, the inner membranes having become soldered at the periphery of the affected zone, though free in the centre.

During life acute serous lepto-meningitis of the vertex of the cerebrum, as seen through the trephine aperture, after reflection of the dura mater, presents the appearance of a clear fluid con-

tained in the almost invisible arachnoid meshwork, with many of the minute arteries lifted from the pia mater as if floated in the fluid itself, but probably resting on the outer strands of the œdematous pia mater. The arteries appear far more numerous than is usually represented. The veins are very large and full. The pia has a swollen œdematous look and is much injected. The underlying brain is moist and glistening, the white substance especially so. The interval between the surface of the brain and the dura in these cases is variable, one eighth to one quarter of an inch, and in one instance of acute external hydrocephalus, where the superficial pressure was great, it measured one inch.

In the instance alluded to, the patient was dying from acute serous lepto-meningitis (probably, however, of tubercular origin), with all the more marked systems of pressure on the brain:—coma, stertorous breathing with lividity of face, widely dilated and fixed pupils. A disc of bone was removed from the skull, and on reflecting the dura mater some meshes of arachnoid protruded, forming a hernia of the membranes filled with fluid. On puncturing these, the fluid at first escaped in a jet which projected for about five inches upwards and outwards from the aperture. It was clear cerebro-spinal fluid, probably a little thinner than usual. Ten ounces were removed. The brain was depressed for fully an inch within the dura mater. Before the end of the operation it had increased in volume, so that it was raised for at least one half the distance, and in twenty-four hours it had reached the internal surface of the dura. The serious symptoms all vanished, and consciousness was temporarily restored.

In all cases of serous lepto-meningitis the appearances after death are greatly altered, and are often disappointing, as in many instances it is difficult to find by the naked eye, appearances sufficient to explain the fatal termination. This may be accounted for by the fact, that the mode of opening the head by the removal of the calvarium is apt to destroy, or cut into the arachnoid tissue, and so permit the escape of serum. As there are few capillaries in the meninges, it is the small superficial veins that are most injected. Even the congestion of the white substance, seen during life, disappears to a very great extent owing to post-mortem changes. These changes have been described as a kind of cerebral post-mortem rigidity or coagulation, which compresses the capillaries and empties them of their contents. The only post-mortem indications of hyperæmia of the white

matter are the distension of the venules, and the oozing of small drops of blood from their cut mouths, on section of the brain.

Purulent lepto-meningitis.—In purulent meningitis, which may be embolic, besides the hyperæmia and serous exudation, there is great exudation of leucocytes into the perivascular spaces with intense dilatation of the veins, their periphery being marked with opaque white and yellow exudation, caused by the leucocytal exudation and its degeneration into pus. The coats of the vessels are traversed by leucocytes, and the veins are frequently plugged with granular coagula. The exudation is at first confined to the arachnoid tissue and pia matral clefts and spaces. It may extend to the brain, and through the transverse fissures to the *telæ choroideæ*, which then swell and become covered with pus and fibrino-purulent exudation. The underlying brain substance is then moist and soft, and the fluid within the ventricles is turbid and sero-purulent.[1]

When the base of the brain and of the cerebellum is affected with purulent lepto-meningitis, the spinal axis is covered with pus, which may be so abundant as to reach into the lowest recesses of the spine, the *cauda equina* being surrounded by and steeped in pus.

It is stated that there are cases in which the purulent acute lepto-meningitis does not produce encephalitis, but in those seen after inflammation due to infective causes, there is always involvement of the cerebral tissue, especially the cortex, which undergoes extensive inflammatory changes. The inflammatory process may extend into the cerebral cortex by the pial sheaths of the veins, which become filled with leucocytes, or there may be direct extension of the inflammation facilitated by prior adhesion of the membranes. In such cases the nerve tissue of the brain and cord is often infiltrated with cells. Accompanying the purulent meningitis there is frequently involvement of the interior of the brain in the form of minute extravasations in the perivascular sheaths and larger ones in the cerebral tissue; the latter being generally surrounded by an area of cerebral softening, which areas may ultimately be the seats of purulent exudation.

[1] These appearances were well seen in one of the cases of cerebellar abscess which had burst into the subdural space and set up purulent lepto-meningitis.

CASE XIV. *Purulent cerebro-spinal lepto-meningitis, with infective thrombosis of sigmoid sinus, originating in otitis media purulenta. Death. Coming under observation on the seventh day of her illness. Duration of illness nine days.*

A. J., female, aged 17 years, admitted into Royal Infirmary on 9th August, 1889, the sixth day of illness, suffering from purulent cerebro-spinal meningitis with thrombosis of sigmoid sinus, originating in infective left otitis media.

History.—*Heredity.*—Nothing definite. *Personal.*—Patient stated that she had been in good health till thirteen years of age, when she had a severe sore throat, after which she became somewhat deaf in the left ear, but there was no otorrhœa. The defect in hearing continued from that time. Eight months ago she had a slight illness, with symptoms of pain in the stomach, vomiting, and vertigo. Three months ago a purulent discharge occurred for the first time, from the left auditory meatus. It came in drops, and had a bad odour.

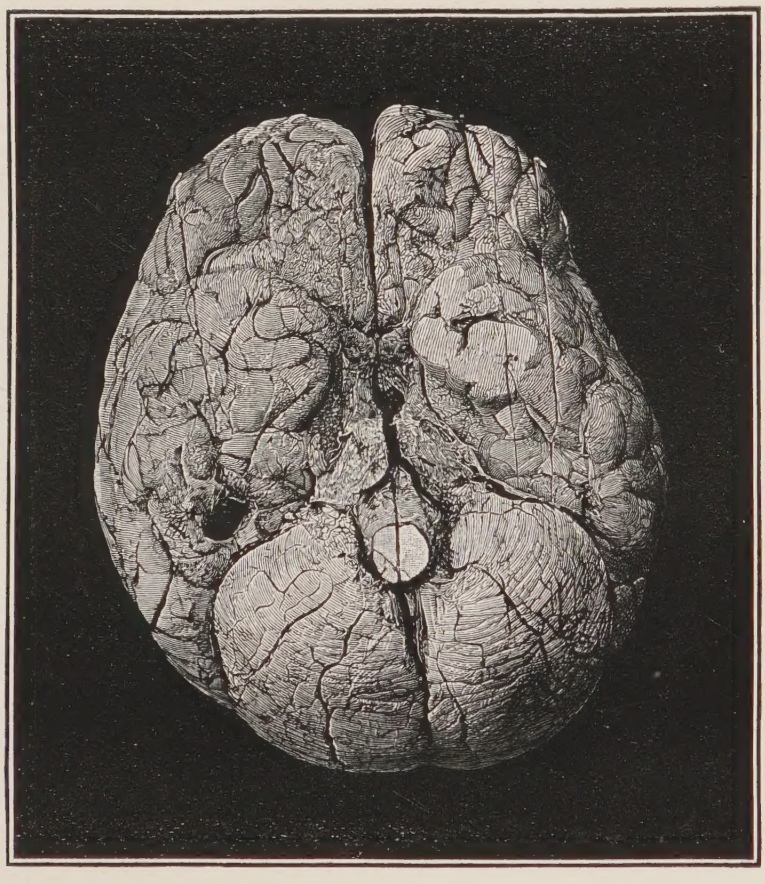
Present Illness.—On 3rd August (six days before admission) she experienced pain in the left ear, which gradually increased in severity and extent. On the following day it radiated all over the left side of head, being especially severe just above the ear. On the third day the pain in the head was even greater, and besides she experienced pain in the region of the stomach, and several times she vomited. On the same day she had a severe rigor, followed by profuse sweating. Since the third day of her present illness she has vomited constantly, the act being instantaneous, and not preceded by nausea. On the sixth day she had a second rigor similar to the first.

State on Admission.—Patient, who was a bright, fairly-developed girl, with a pale, anxious, pain-stricken face, answered questions promptly and with ready intelligence, complained of pain over the left parietal region, especially behind the left ear, which, though persistent, occurred with agonizing exacerbations, causing her to screech out. Percussion over this area intensified the pain.

There was extreme tenderness to touch over the whole left posterior cervical triangle and along the course of the jugular, extending to the base of the neck, though the painful area lessened from above downwards. The tissues over this area were swollen and thickened (tumor albus). Besides, she suffered from pain over the lower part of the chest and abdomen, which starting from the back and sides, travelled toward the front. These pains occurred at various levels, but she could not exactly liken them to girdle pains. It is probable that the severe intracranial pain masked that in the trunk. There was herpes in the left corner of the mouth and an anomalous rash of a diffuse dusky-brown colour over the left side of the neck and upper portion of the chest, especially on the left side.

Motor Functions.—She lay almost motionless on her back, with very little, if any, retraction of the head, seldom moving her limbs or making any voluntary effort, though frequently desiring to be slightly turned in bed, She seemed to suffer from extreme prostration. The movements of her limbs

FIG. 38.—ABSCESS IN RIGHT TEMPORO-SPHENOIDAL LOBE.



Base of brain showing abscess in right temporo-sphenoidal lobe, which communicated through softened membranes with an erosion in tegmen tympani.
From photograph. This specimen was given to author by Dr. Erskine, Glasgow.

To face page 88.

when put to the test, were extremely slow and feeble. Her grasp was lost. She could not, with either hand, make the slightest impression on the dynamometer: her lower limbs could be slowly dragged a little way upwards. This muscular prostration was a marked feature.

Sensory Functions.—There was hyperæsthesia over the anterior abdominal wall, but with this exception the sensation all over the body was blunted, that of touch being correct, though very dull. The sensation of pain was almost gone, except over the left side of head, which was acutely sensitive. The cutaneous reflexes of both sides were blunted. Patellar reflex slight, and ankle clonus absent. She had complete control over bladder and bowels.

Special Functions.—Eyesight, which used to be good, became defective four days previous to admission and continued so since. She was unable to read even the largest print. She had occasional diplopia, though there was neither strabismus nor nystagmus apparent. She had marked photophobia. The pupils were in a state of stabile myosis.

Ophthalmoscopic Examination.—Both discs were hyperæmic, veins distended. There was a slight perivascular exudation. These appearances were suggestive of commencing optic neuritis. On 10th August, optic neuritis distinct in right eye.

Hearing.—On the right side she heard a watch at five inches; the left ear was absolutely deaf, as tested by both aerial and bone conduction, left membrana tympani gone, atresia of external auditory canal.

Taste and smell. Normal.

Temperature, pulse, and respiration all high (see Chart).

Operation.—This was evidently a case in which there was infective absorption from the otitis media, affecting the sigmoid sinus, and there was a suspicion, from the persistent pains at various levels of the thorax and abdomen, of involvement of the cord. It was considered hopeless, but the mastoid antrum was exposed along with the sigmoid sinus and the dura mater of the posterior fossa for half an inch beyond. In both of these situations putrid pus was found, in the latter intra- and extra-dural, and from the posterior fossa of the skull it continued to ooze. The left sigmoid sinus was ablated. This operation did not relieve the patient, whose symptoms persisted, very much as have been described, until her death on 11th August, at 3.30 a.m., having been under observation about forty-eight hours.

Post-Mortem Examination.—The body was well nourished, and post-mortem rigidity was marked. The skin of the abdomen was slightly mottled, and that of the left side of neck and shoulder markedly so, the spots being of a purple brown colour. The left sigmoid sinus was exposed, through the artificial aperture in the mastoid, which contained dressing and was aseptic. The heart, liver, kidneys and spleen presented no abnormal naked eye appearances. The mesenteric veins were turgid, and the intestines congested. The right lung was adherent to the chest wall over its upper half. The pleuritic adhesions were loose and easily broken down. They were recent. There was slight hypostatic congestion.

Brain and Spinal Cord.—The spinal cord was removed by excision of the bodies of the vertebræ, from the fourth lumbar to the third dorsal inclusive. On incising the intervertebral disc, between the fifth and fourth lumbar, the knife penetrated the dura mater of the cord at the cauda equina, when two drachms of yellow fluid pus escaped. A further escape of pus to the extent of one drachm occurred about the ninth dorsal vertebra, the chisel having penetrated the dura of the cord at that point. There was no pus in the peridural space of the canal, though the

CHART A.—PURULENT CEREBRO-SPINAL LEPTO-MENINGITIS.

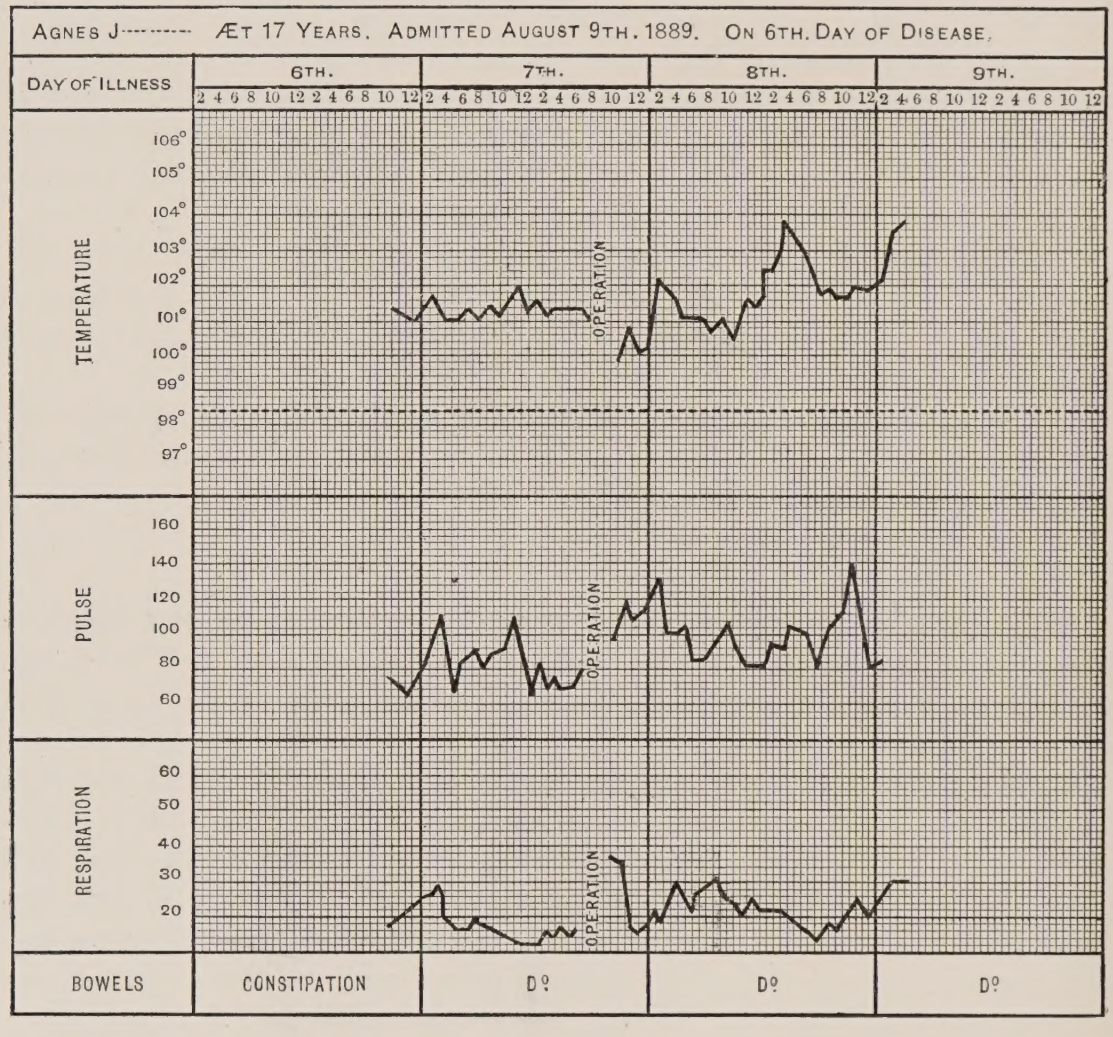


Chart of temperature, pulse, and respiration in a case of purulent cerebro-spinal lepto-meningitis and thrombosis of sigmoid sinus arising from left infective otitis media.—Death.

external surface of the dura was congested. On the dura being divided longitudinally, along the anterior surface of the cord, purulent exudation was found bathing the cord, nerve roots, and pia mater. Throughout its whole extent, membranous-like patches were found where the pus was confined within fibrinous meshes. Between these fibrino-purulent layers, fluid yellow pus was collected in irregular cavities. On the pus being examined microscopically, it was found teeming with micro-organisms,—bacilli and micrococci,—no doubt partially due to post-mortem changes. The veins of the pia mater were filled with dark blood, appearing congested and very tortuous;

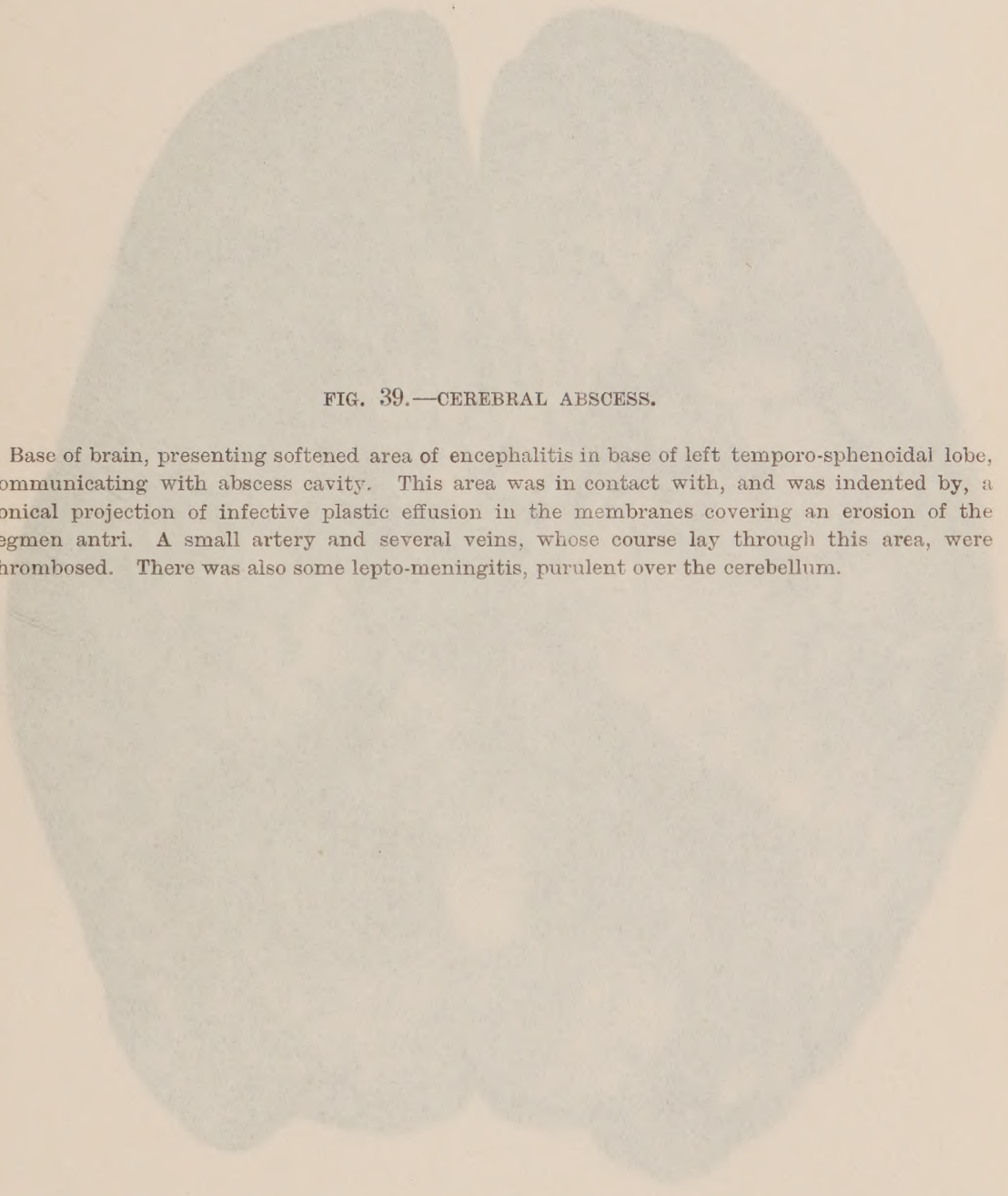


FIG. 39.—CEREBRAL ABSCESS.

Base of brain, presenting softened area of encephalitis in base of left temporo-sphenoidal lobe, communicating with abscess cavity. This area was in contact with, and was indented by, a conical projection of infective plastic effusion in the membranes covering an erosion of the tegmen antri. A small artery and several veins, whose course lay through this area, were thrombosed. There was also some lepto-meningitis, purulent over the cerebellum.

To face page 90.

Brain and Spinal Cord.—The spinal cord was removed by excision of the bodies of the vertebræ, from the fourth lumbar to the third dorsal inclusive. On incising the intervertebral disc, between the fifth and fourth lumbar, the knife penetrated the dura mater of the cord at the cauda equina, when two drachms of yellow fluid pus escaped. A further escape of pus to the extent of one drachm occurred about the third dorsal vertebra, the chisel having penetrated the dura of the cord at that point. There was no pus in the peridural space of the canal, though the

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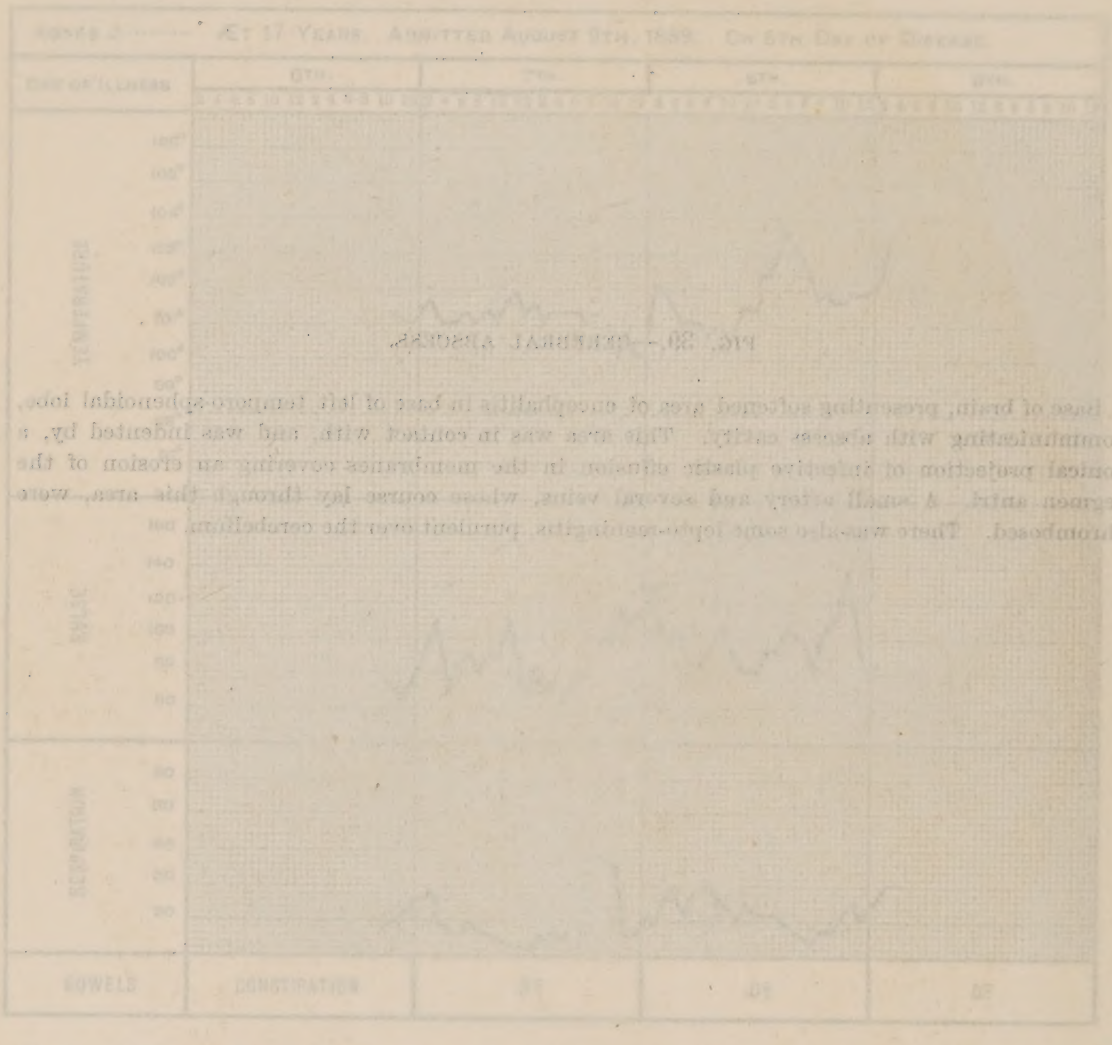
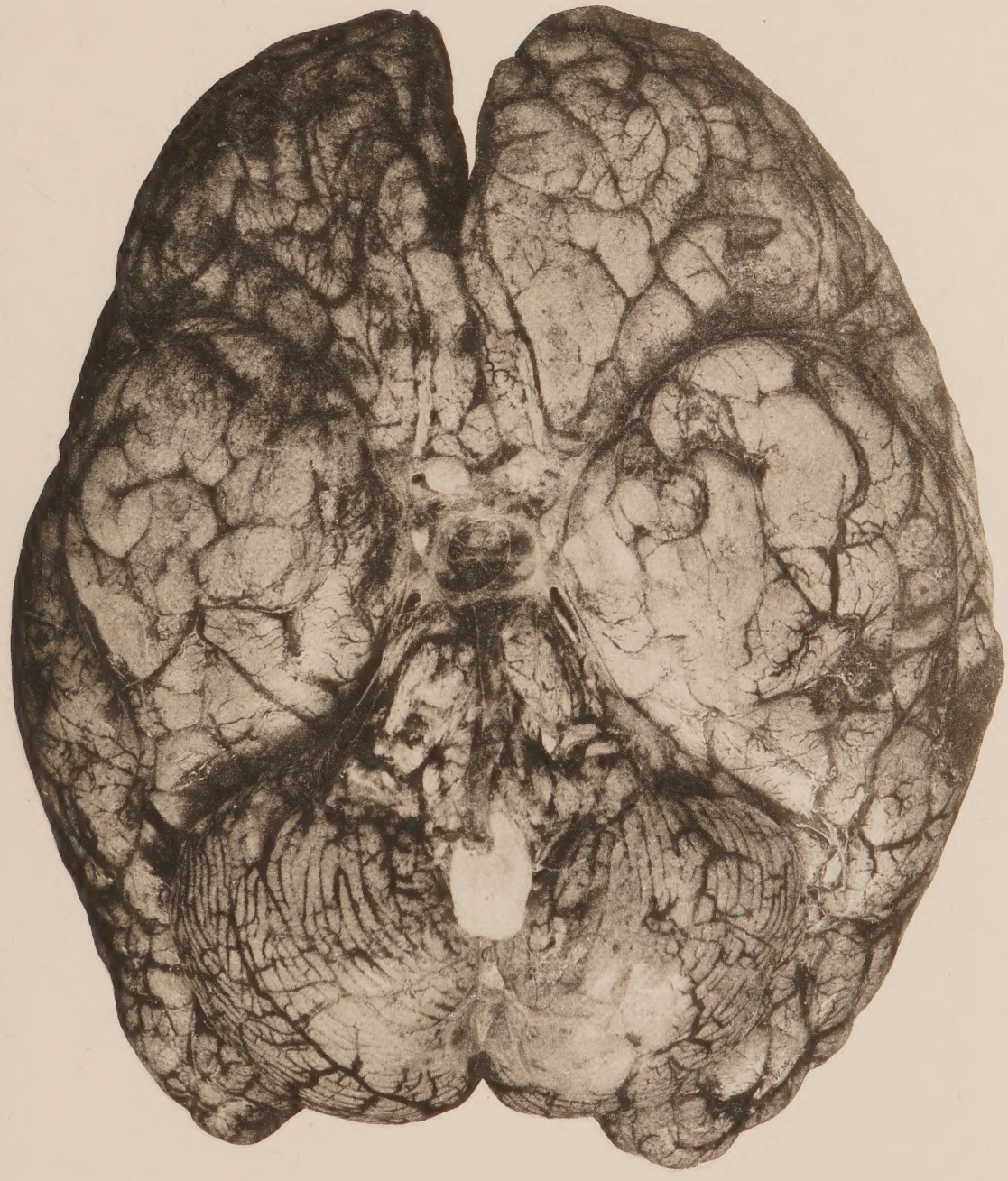


Chart of temperature, pulse, and respiration in a case of purulent cerebro-spinal lepto-meningitis and thrombosis of sigmoid sinus arising from left infective otitis media.—Death.

external surface of the dura was congested. On the dura being divided longitudinally, along the anterior surface of the cord, purulent exudation was found bathing the cord, nerve roots, and pia mater. Throughout its whole extent, membranous-like patches were found where the pus was confined within fibrinous meshes. Between these fibrino-purulent layers, fluid yellow pus was collected in irregular cavities. On the pus being examined microscopically, it was found teeming with micro-organisms,—bacilli and micrococci,—no doubt partially due to post-mortem changes. The veins of the pia mater were filled with dark blood, appearing congested and very tortuous.

FIG 39.



the cord substance was unusually soft. *On Histological Examination* of the cord and membranes being made, it was found that there was a thick layer of pus between the membranes and the cord, and also an inflammatory purulent exudation in the membranes themselves, many of the vessels were thrombosed, the thrombi being recent. There were also extravasations of red blood corpuscles at some points.

Brain.—The cerebral dura mater was normal; the pia mater was thickened over the vertex and parietal bones. All the cerebral blood-vessels were congested. There was a patch of plastic exudation about the posterior part of the circle of Willis. On the cerebellum being removed it was seen that the dura clothing the left cerebellar fossa was covered by a layer of yellow inspissated pus. This layer was most dense on the posterior aspect of the left petrous bone, especially over the sigmoid sinus. From this point the pus spread backwards and inwards over the cerebellar dura mater to the internal aspect of the right side of the foramen magnum, over the margin of which it was traced into the spinal canal. The thin layer of pus occupying the inner, lower and posterior third of the right cerebellar fossa was continuous with the layer in the left cerebellar fossa, and with that in the spinal canal, and was evidently an extension from the left side. It was also clear that the pus had originated in and around the left sigmoid sinus, which contained a disintegrating purulent thrombus, and the suppuration had spread from it into the cerebellar fossa and spinal canal. The pus was chiefly formed into a tough layer, capable of being stripped off, though there was besides a small quantity of liquid purulent secretion without any offensive odour, principally situated about the sigmoid sinus.

The right lateral and sigmoid sinuses were free from disease. The pia mater covering the cerebellum was congested generally, but markedly so over the lower and posterior aspects of the left hemisphere, corresponding to the purulent layer lying on the dura. Over the greater part of the right hemisphere and the upper surfaces of both hemispheres, the congestion of the pia mater was comparatively slight. The surface of the cerebellum itself presented no abnormal naked eye appearance. There was no abscess in cerebrum or cerebellum. The left petrous and occipital bones, including the foramen magnum as well as upper three cervical vertebræ, with the soft parts of the neck attached, were removed for dissection. On examining them, it was found that on the outside of the skull the vessels and nerves were all fused together, along with many enlarged glands, formed in strings extending into the neck from the suboccipital and submastoid region. The size of the glands varied from a pea to that of a walnut. The thrombosis of the left sigmoid sinus did not extend beyond the skull; the jugular vein in the jugular fossa was occluded with firm clot, and below this point it was free from clot, although its coats were congested and somewhat thickened.

Remarks.—Judging from the erosions in the temporal bone, the infective disease had evidently spread from the mastoid antrum and middle ear to the sigmoid groove and sinus. The perineural sheaths of the seventh and eighth nerves participated in the purulent meningitis, and pus was formed in the internal auditory meatus, but the internal ear was free from erosion.

CASE XV. *Cerebro-spinal lepto-meningitis of infective origin, perforation of tegmen tympani, extradural abscess over tegmen arising from chronic otitis media. Death. Coming under observation on the sixtieth day of illness, dating from subacute symptoms. Duration of illness, sixty-six days, the acute symptoms lasting eleven days.*

D. F., aged 24 years, a commission agent, was admitted into Royal Infirmary on 5th December, 1889, suffering from meningitis, arising from infective otitis media, with extensive caries of the tympanum, the cavity of which was filled with granulation masses protruding into the external auditory meatus.

History.—*Heredity.*—Family history was good. A brother had had a discharge from one of his ears which lasted only a few months. *Personal.*—Patient was always able for his work, and was considered healthy, though from an early age he had had a right otorrhœa.

Present Illness.—Two months previous to admission he complained of pain in the right ear, with occasional giddiness. At that time he was examined by an aurist, who found granulation tissue protruding into the right external auditory meatus. Hearing by aerial conduction was lost, while the vibrations of the tuning fork were heard by bone conduction. He was advised to drop into the external ear a 50 per cent. solution of rectified spirit in water. A few days subsequently he became unwell, and was confined to bed, complaining of pain in the right ear, head, and abdomen, and when he stood upright he became giddy and vomited. This attack lasted about ten days. When he recovered, he applied again to the aurist, who, on the 27th November, 1889, removed by Wilde's snare a mass of granulation tissue. Subsequently the pain increased in the ear and head, and on the 2nd December, the fifth day after the aurist's operation, he felt cold and shivered, although on the previous day he looked flushed and feverish. On this same day the aurist removed two more growths from the middle ear. All these growths were attached to the upper and posterior wall of the middle ear. On the following day his symptoms were increased; the pain was very much aggravated and extended to the brow and vertex. He was restless, thirsty, with quick pulse and high temperature, 103° F. to 104° F. (axillary), and he was slightly delirious.

State on Admission.—When admitted, about the sixtieth day of his illness, patient, who had a flushed face, lay quietly in bed, with his eyes closed, though he was not asleep. His intelligence was clear, his cerebration was rather over-active. He was fretful under examination. He complained of intense pain extending all over the head, though most severe over the right ear and back of neck. He had also pain in his spine, and several times voluntarily drew attention to this spinal pain. On inquiry it was found that he had complained of pain in his spine in November and December during his previous attacks. There was no swelling over the course of the internal jugular, though there was tenderness on pressure, equal however on both sides. With the exception of a slight facial paralysis on the right side, there were no motor symptoms. The superficial sensations were hyperacute,

FIG. 40.—ABSCESS IN TEMPORO-SPHENOIDAL LOBE (SEEN IN SECTION).

Section of Brain, showing abscess in temporo-sphenoidal lobe with its exit aperture at base.

Follows page 80.

the reflexes were normal. He vomited several times during the first examination, which lasted half an hour. His pulse was quick, weak, and irregular. His temperature was 104° F. His urine was albuminous.

Examination of Ears.—On the right side he was deaf to whispers, but heard the raised voice. By bone conduction he heard the tuning fork but not the watch. The membrana tympani was gone, and the cavity was filled with granulation tissue which was seen after the removal of the inspissated pus. The left ear was normal.

Examination of the Eyes.—The movements of the ocular muscles were perfect. The fundus of the left eye showed slight optic neuritis, and the disc was hazy. The disc of the right eye on its inner, upper, and lower aspects was slightly obscured, but the vessels were not prominent. There was no retinal exudation.

During the first two days subsequent to admission patient was very restless and irritable, tossing about from side to side, complaining of thirst, pain all over the head, back of neck, and in the right ear, occasionally in both ears.

The diagnosis arrived at was lepto-meningitis of the brain and spinal cord, arising from infective otitis media. There were no indications of abscess of the brain, and none of sigmoid sinus thrombosis, and if abscess were present, it was altogether masked by the prevailing meningitis. It was probable that the granulation masses formed in the middle ear were connected with the tegmen tympani, and if so possibly were protrusions from the dura mater. It was necessary to remove them and the fœtid pus which surrounded them.

Operation.—On 7th December, 1889 (on the sixty-second day of present illness) the mastoid antrum and the cavity of the tympanum were exposed. They were both filled with granulation masses and inspissated fœtid pus. The tegmen tympani was eroded, and a mass of granulation attached to the dura mater protruded through the aperture in the bone into the cavity of the tympanum. On this mass of granulation tissue being pressed aside pus oozed from the cranial cavity to the extent of nearly two drachms. It had been collected between the dura and the skull, and confined there by the granulation tissue, which, springing from the dura, protruded into the cavity of the tympanum, filling the osseous aperture like a plug. The cavities of the antrum and tympanum were so much enlarged by caries as to form one large space without the limiting neck forming the constricting passage. The facial canal was exposed by erosion. All the ossicles had disappeared except the malleus, the head of which was the seat of caries. The whole cavity was rendered aseptic, and filled with antiseptic dressing.

In the succeeding days no improvement followed this operation. Tingling sensations occurred in the finger tips. He had difficulty in micturition, and both sides of his face were covered with perspiration. He slept little, and what sleep he did get was disturbed by dreams. Delirium when awake appeared on the sixty-fourth day of his illness. On the sixty-fifth day he had a jaundiced tinge all over the body, the conjunctiva being markedly coloured. There was marked rigidity in the muscles of the neck, both arms, and both legs. This

rigidity became more pronounced on attempted movement. The twitchings of the fingers and toes became marked, and soon extended to the muscles over the body generally, though the various groups of muscles were erratically affected, the spasms being confined to one limb for five minutes, then ceasing in it and passing on to the other. All the reflexes became exaggerated; touching the soles of the feet caused violent jerks of the whole body. Temporary strabismus was frequent. The pupils, which had remained equal for the first few days, gradually changed, the right becoming slightly larger than the left. Hiccough, which occurred several times in short snatches on the sixty-third and sixty-fourth days, now became continuous and pronounced from the sixty-fifth day. On the sixty-sixth day of his illness, and the fifteenth of the acute cerebral symptoms, he became comatose, in which state he died. Tracings of pulse, respiration, and temperature are given below.

CHART B.—PURULENT CEREBRO-SPINAL LEPTO-MENINGITIS.

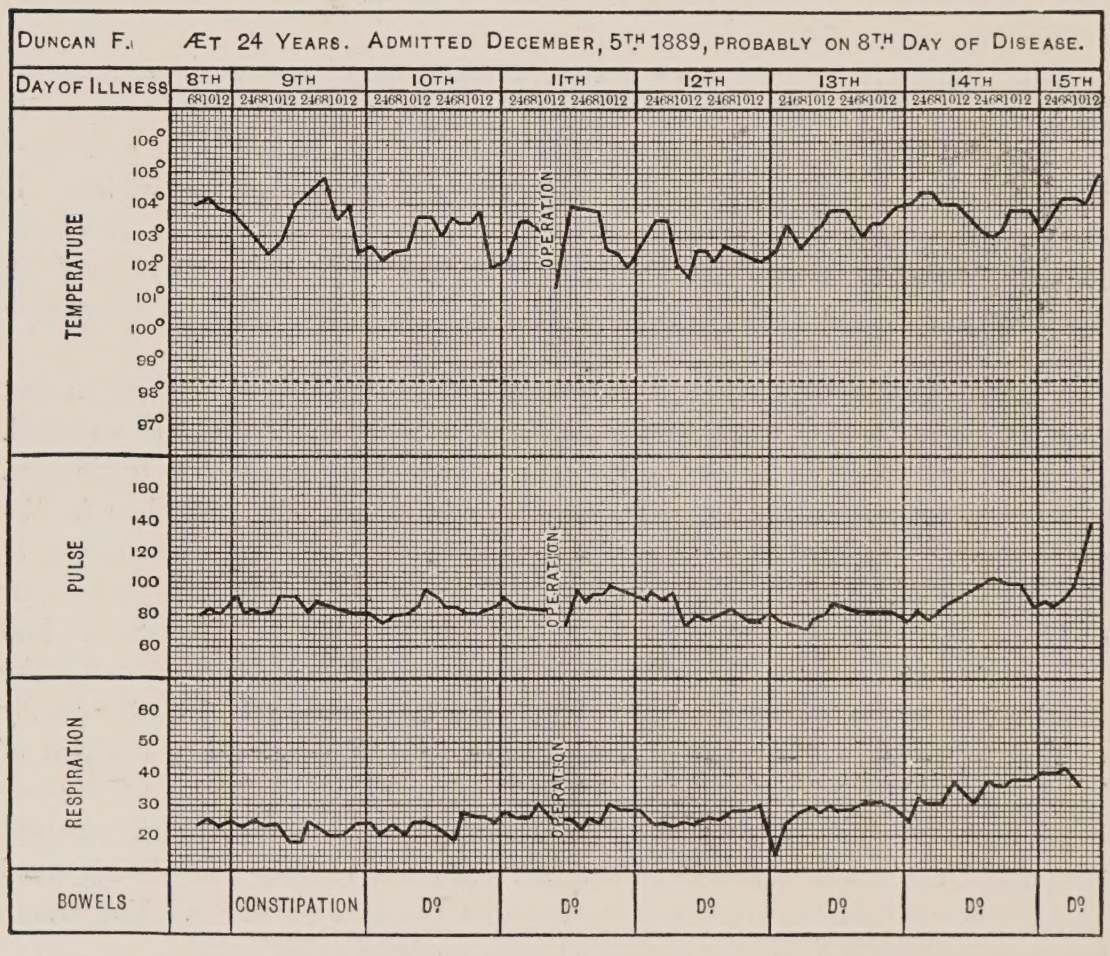


Chart of suppurative cerebro-spinal lepto-meningitis of brain and cord with extradural abscess above tegmen tympani arising from infective otitis media (right). Temperature, pulse, and respirations taken every two hours.

Post-Mortem Examination.—There was a patch of granulation tissue on infiltrated dura mater which could be covered by a halfpenny, situated over the superior aspect of the petrous bone, and communicating with the perforation on the roof of the tympanum. Round this patch there was an inflamed area of the dura mater. Between the sigmoid and lateral sinuses

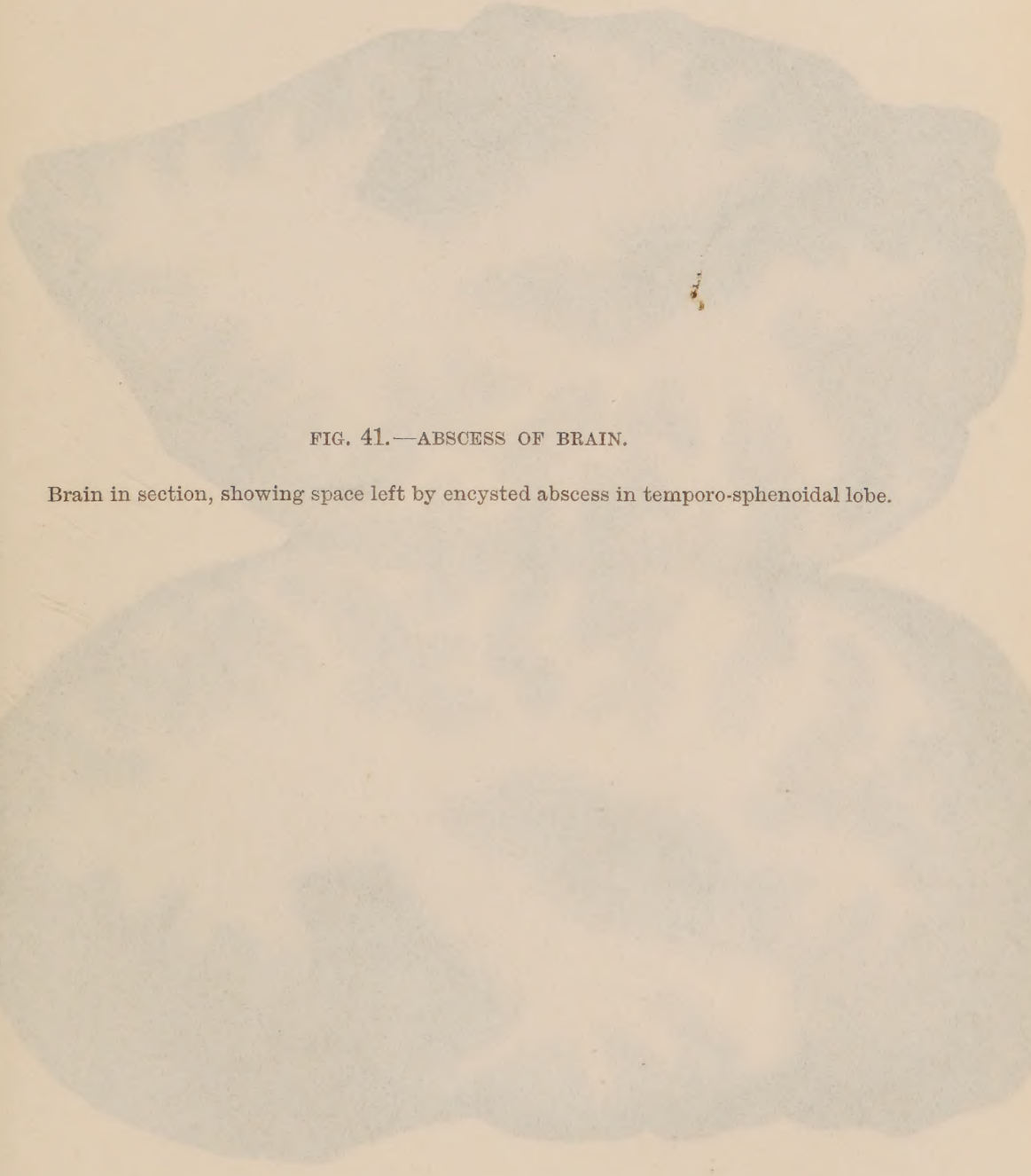


FIG. 41.—ABSCESS OF BRAIN.

Brain in section, showing space left by encysted abscess in temporo-sphenoidal lobe.

To face page 94

rigidity became more pronounced on attempted movement. The twitchings of the fingers and toes became marked, and soon extended to the muscles over the body generally, though the various groups of muscles were erratically affected, the spasms being confined to one limb for five minutes, then ceasing in it and passing on to the other. All the reflexes became exaggerated; touching the soles of the feet caused violent jerks of the whole body. Temporary strabismus was frequent. The pupils, which had remained equal for the first few days, gradually changed, the right becoming slightly larger than the left. Hiccough, which occurred several times in short attacks on the sixty-third and sixty-fourth days, soon became continuous, and commenced from the sixty-fifth day. On the sixty-sixth day of his illness, and the fifteenth of the acute cerebral symptoms, he became comatose, in which state he died. Tracings of pulse, respiration, and temperature are given below.

CHART B.—SUBACUTE CEREBRO-SPINAL LEPTO-MENINGITIS.

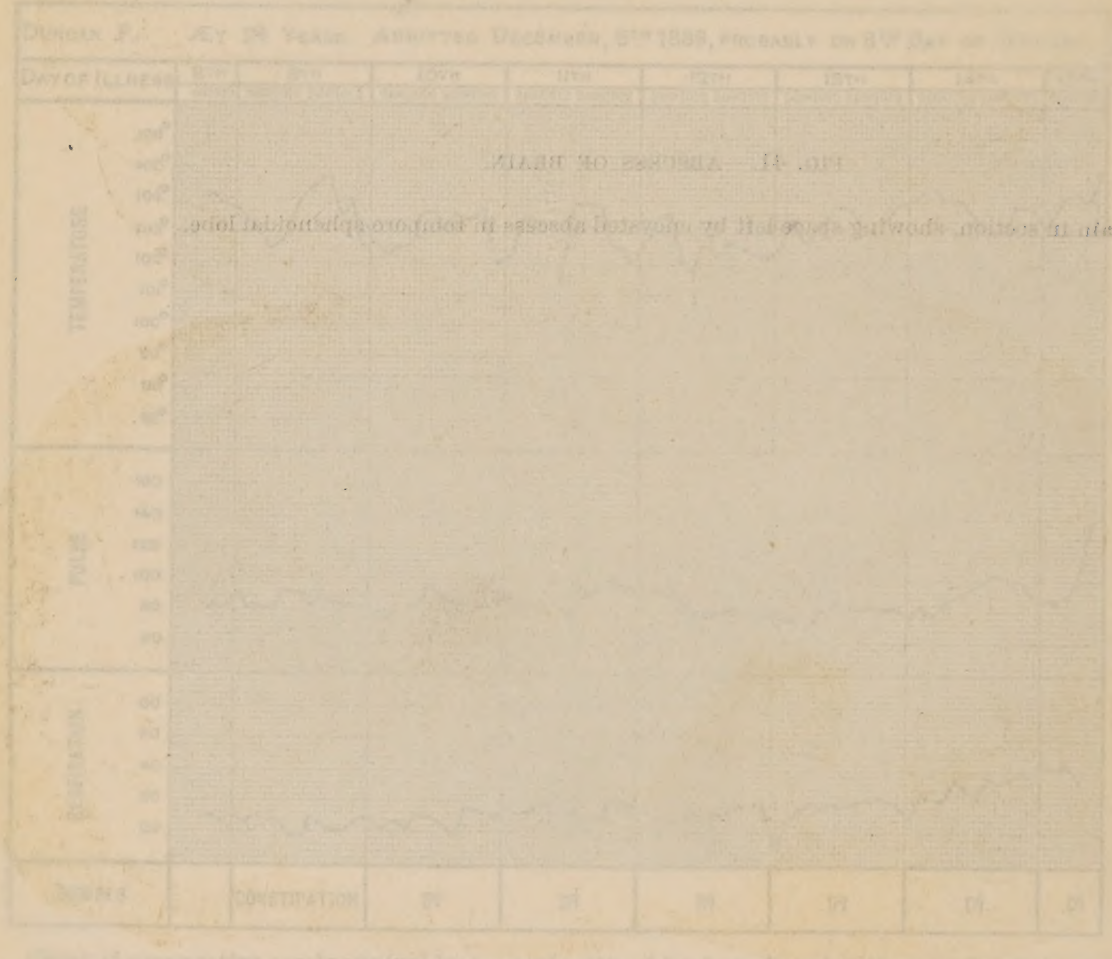


Chart of suppurative cerebro-spinal lepto-meningitis of brain and cord with extradural abscess (left) and cerebellar tropical ataxia from infective otitis media (right). Temperature, pulse, and respiration taken every two hours.

Post-Mortem Examination.—There was a patch of granulation tissue on the inner surface of the petrous bone, and communicating with the perforation in the roof of the tympanum. Round this patch there was an adhesion of the dura mater. Between the sigmoid and lateral sinuses

FIG 41.

and this patch, there was a zone of healthy dura mater. The outer margin of this patch was bounded by the posterior branch of the middle meningeal artery. At this point the inner membranes were both adherent to the dura. There was intense lepto-meningitis distributed over both sides of the brain, base and vertex, and extending to the cerebellum, medulla, and cord. At several points—the anterior aspect of the Sylvian fissure, the flocculus on both sides, the lateral aspects of the cerebellum, and various parts on the vertex—there were patches of plastic effusion yellowish-white in colour, adhering to the arachnoid. Similar but smaller flakes were seen at several places on the cord. Though the lepto-meningitis had extended generally over the cord, it was principally marked at the lower portion of the cervical enlargement and in the dorsal regions. The whole cord was deeply injected, the veins were very prominent and were covered by plastic exudation. When the cerebral membranes were removed a large quantity of cerebro-spinal fluid escaped. On the brain being opened it was seen that there was marked internal meningitis, the interior of the ventricles were intensely injected, the choroid plexuses being covered by greyish-yellow inflammatory effusion. The membranes of the third and fourth ventricles were similarly affected, and the cerebral substance lining all the ventricles was softened and pulpy. In examining the bones at the base of the skull it was seen that the perforation in the roof of the tympanum was situated to the outer and anterior aspect of the superior semicircular canal, its inner border corresponding to the inner side of the tegmen tympani. It measured in diameter one quarter of an inch both antero-posteriorly and from without inwards. About two-thirds of the gap lay over the mastoid antrum, while one-third lay directly over the tympanic cavity. The sigmoid sinus was free, though the wall seemed a little thickened towards the sigmoid groove.

Remarks.—The meningitis originated in the infective otitis media. The removal of the granulation masses projecting in polypoid form into the tympanic cavity, probably acted as the exciting cause. Some of these masses sprang from the external surface of the dura mater. The wound formed by their abruption, left in contact with the infective matter contained in the interstices of the middle ear and mastoid cells, was a source of danger. The intricate passages of the middle ear, the mastoid antrum and cells cannot satisfactorily be rendered thoroughly aseptic in such a case by applications through the external auditory meatus. Free exposure of the antrum and tympanic cavity from behind the ear enables the granulation masses to be fully exposed to view, when they may be thoroughly removed and the cavity rendered aseptic. Of course meningitis may arise in such cases even when they have been left to run their course without treatment of any kind, and also in such as have been treated by simple cleansing of the tympanum from the external auditory meatus.

CASE XVI. *Infective purulent cerebro-spinal lepto-meningitis, with thrombosis of the sigmoid sinus from right otitis media purulenta. Coming under observation on fifteenth day of disease. Duration of illness seventeen days. Death.*

J. M'R., aged 23, ironmoulder, admitted to Royal Infirmary on 25th September, 1890, suffering from suppurative cerebro-spinal lepto-meningitis, thrombosis of the sigmoid sinus, and small cerebral abscess in temporo-sphenoidal lobe, arising from otitis media purulenta.

History.—*Heredity.*—Nothing definite in family history. *Personal.*—With the exception of otorrhœa after measles in childhood, he has been in fair health.

Present Illness.—A fortnight before admission, he became ill, and was confined to bed; complaining of pain chiefly in the occipital region, though also in the right shoulder, and over both sides of the trunk and abdomen. Four days before admission, he had a convulsion in which he became rigid, and was unconscious for about twenty minutes. Some hours after the fit, he vomited repeatedly. He had had several rigors during his illness. The precise dates on which they occurred cannot be specified. He had been treated by a medical man for "a cold" during the ten days before coming into hospital.

Condition on Admission (on fifteenth day of illness).—Patient was semi-conscious, but could not be roused to give rational answers. The mouth was rather drawn to the left, as if there was a slight shade of facial paralysis on the right side. The pupils were small, and did not react to light. The ocular muscles were not paralysed; there was constantly oscillating nystagmus. The arms and legs moved, but he could not be roused to put forth any effort; his hand closed over the dynamometer, but exerted no pressure.

Reflexes.—Plantar reflexes slight and equal, knee jerks equal and normal, no ankle clonus, no epigastric nor abdominal reflexes.

He had evident pain on the right side of the head, which was increased on tapping, especially over the mastoid. The head was retracted and rigid, and when moved, he cried out. Over the upper third of the right internal jugular, there was tenderness on pressure; there was slight submastoid œdema.

Ears.—There was a large perforation in the right membrana tympani, and dried pus in the meatus. The left membrane was normal.

Eyes.—The fundi, owing to nystagmus and fretfulness of patient, could not be examined.

He had a mitral systolic murmur, and some bronchial râles in the lungs, especially over the bases. His temperature was 104° F., pulse 89, respiration 19. The urine which was withdrawn by catheter was albuminous. He had slight cough.

It was evident that the patient was suffering from suppurative cerebro-spinal lepto-meningitis.

FIG. 42.—CEREBELLAR ABSCESS AND PURULENT ENCEPHALITIS SURROUNDING VENTRICLES.

Hardened brain and cerebellum in section, from a case of cerebellar abscess and extensive purulent lepto-meningitis, with encephalitis and cerebral softening surrounding the ventricles, the cavities of which were filled with pus. The lateral ventricle, its descending horn, and a portion of its posterior horn, are seen in section.

Follows page 80.

Operation (25th September, 1890).—The mastoid antrum was opened and found filled with pus. The sigmoid sinus was exposed for about an inch, and it was covered by a thick layer of purulent exudation. The dura was opened on the outer and posterior aspect of sinus, and some pus escaped from the inner membranes in drops.

In the membranes over the base of the temporo-sphenoidal lobe, there was a small collection of pus communicating with an ulcerated patch in the dura mater, over the tegmen tympani. From each of the two apertures, the sigmoid groove and tegmen tympani, there was a constant oozing of pus, which continued to well out even after extensive antiseptic washings. It was evident that there was a diffuse, suppurative, lepto-meningitis in the middle and posterior fossæ; the pus from which was gradually finding an exit by the apertures in the skull.

During the short time patient lived after operation, his condition was very much the same. The cough developed, and he had prune juice expectoration. The head and neck remained retracted. He had slight deviation of both eyes towards the right. He was in a condition of noisy delirium, alternating with periods of complete unconsciousness, and he died forty-two hours after operation, on seventeenth day of illness.

Post-Mortem Examination.—The spinal canal was first opened. The whole of the subdural space was filled with pus as far as the cauda equina. On the skull being opened, the dura mater was found more adherent than was natural. There was a diffuse, internal, and external lepto-meningitis which was suppurative at the base, especially over the basi-sphenoid, and there appeared to be a communication between this pus and that in the third ventricle. The walls of the latter were soft, creamy, and diffluent. The subdural pus on the basi-occipital communicated with the pus in the subdural space in the cord, so that the pus found in the latter had probably partly gravitated from the cranial cavity, though no doubt the greater part arose from infective inflammation of the spinal membranes.

The right temporo-sphenoidal lobe was very soft. The small intra-membranous abscess in connection with the superficial ulceration at its base over the tegmen tympani had been thoroughly evacuated, and was now collapsed, containing only a small quantity of blood.

There was no pus outside the dura mater except over the tegmen tympani; and in right cerebellar fossa near the petrous bone. The right sigmoid sinus was thrombosed—the walls being thickened, and the sinus occluded. There was no pus round the sinus, as it had been removed at operation. Both lungs were in a state of deep congestion. The left lung was slightly emphysematous along its anterior margin. There was a calcareous mass in the apex of the left lung; other organs normal.

Remarks.—It appears that the lepto-meningitis in this case had arisen from the chronic otitis media, and that it was the chief lesion, extending over the whole brain and spinal cord. The intra-membranous abscess at the base of the temporo-sphenoidal lobe was very small, and although it is possible that the pus which it contained had burst into the subdural space, and had set up the suppurative lepto-meningitis, yet it is probable that the

pathogenic organisms had disseminated themselves into the subarachnoid and subdural space, setting up the suppurative cerebro-spinal meningitis before the local inflammatory process at the perforated tegmen and dura had time to effect the complete soldering of the membranes, and that the small intra-membranous abscess was only a part of the general basal involvement, the pus having formed more rapidly at the point of origin. The death was clearly attributable to the suppurative lepto-meningitis.

The late period at which the patient came under observation prevented many of the symptoms, which no doubt previously manifested themselves, from being detected; but the pains which he suffered over both sides of the trunk, the retraction and rigidity of the head, the high temperature, the lack of power over the extremities, all pointed to serious cerebro-spinal meningitis. The insidiousness of the affection was manifest by the fact that he had been attended for a week prior to admission by a well-informed practitioner, who could only attribute his illness to a severe cold. The patient and his friends had not mentioned the ear disease to him. This case bears a certain resemblance to Case No. XV.

There were here two points inside of the cranium from which the pus emanated, the sigmoid groove as well as the tegmen tympani. In this instance the lepto-meningitis had first been produced at and spread from the eroded dura over the tegmen tympani. Had this part, however, been able to resist the attack, the lepto-meningitis would probably have occurred some time later from involvement of the cerebellar fossa by infective extension from the sigmoid sinus.

CASE XVII. *Cerebro-spinal infective suppurative lepto-meningitis, with acute encephalitis of frontal lobe, and diffuse abscess, originating in a compound comminuted depressed fracture of the frontal bone. Death. Post-mortem examination. Coming under observation on twelfth day of illness and twenty-first day after injury. Duration of illness, thirty-six days.*

J. D., aged 7 years, was admitted into Royal Infirmary on 9th June, 1892, on the twelfth day of his illness and on the twenty-first from date of injury, suffering from infective suppurative cerebro-spinal lepto-meningitis, with localized collection of pus in the left frontal lobe, arising from a compound comminuted depressed fracture of the left frontal bone, with injury to dura and cerebrum.

History.—*Heredity.*—No hereditary illness traced. *Personal.*—Hitherto always healthy.

Present Illness.—Three weeks prior to admission patient fell over an embankment, striking his head, a wound being inflicted about an inch in length. He was rendered unconscious for some minutes, after which he was able to walk, and seemed otherwise well. For nine subsequent days he was able to move about, played as usual, and went daily to the doctor to have his wound dressed. During that time the wound was tardy in healing, and a crop of pustules formed in its periphery.

On the ninth day he became so weak that he had to go to bed. He felt sick, and had pain in the back of the head. On the tenth day he had severe diarrhœa, was feverish, thirsty, and had a foul tongue. The wound suppurated freely, and there was a faint reddish blush round it. For the next few days he was dull, languid, and apathetic, but answered questions intelligently. The temperature (axillary) during this period was from 99° F. to a little above 100° F.

Condition on Admission.—When he entered the hospital, on the twelfth day of his illness and three weeks from time of injury, there was a wound about an inch in length, discharging fœtid pus, situated on the hairy scalp, two inches above the right eyebrow and about the same distance from the middle line. This wound was directly continuous with a depressed comminuted fracture of the frontal bone. He was very quiet, but could answer simple questions intelligently ; did not complain of pain except when asked, and then indicated the back of the head as the part affected. He was very feeble, could with difficulty move his arms and legs, and had left brachial paresis. He had double optic neuritis. The right pupil was generally larger than the left.

Operation (10th June, 1892).—The depressed comminuted fracture was elevated by first removing a disc of bone at its periphery. On extracting the fragments a small quantity of pus escaped. A minute fragment of inner table of skull had been driven edgeways through the dura into brain. The dura externally was inflamed and thickened, and covered with granulation tissue. On the dura mater being opened there was an escape of pus from the subdural space and the brain substance, the latter directly under the position of the depressed bone was considerably disintegrated and pulpy. From the disintegrated brain substance pus oozed out to the extent of an ounce. A second trephine opening was made posterior to the first, as there was a continuous oozing of pus from between the membranes in that direction. It gave vent to some drachms of pus, which issued from between the membranes. The exposed brain tissue at once formed into a hernia cerebri, which was indicative of encephalitis. The whole of the exposed tissues were washed, and an attempt made to render them aseptic. The parts were dressed. The pus which was removed was found to contain the streptococcus pyogenes and staphylococcus aureus, and as the membranes were seriously involved the prognosis was very grave.

Subsequent Course.—Patient seemed relieved to an extent by the operation, but it was clear that the meningeal symptoms still persisted. There was occasional squint, flushings of the face, muscular tremors, and flitting paralysis of various members, chiefly on the opposite side of the body, but latterly on both sides. Occasionally he had fits of rigidity all over the body. He was extremely feeble, and either could not or would not put forth any muscular effort. Both motions and urine were passed in bed. His temperature remained slightly above normal for first fortnight, reaching occasionally 102° F., and then increased on the 29th day of his illness to 103° F., from which time it continued high until his death on 4th July. (See Temperature Charts.) His emaciation during that period was a marked feature.

Post-Mortem (6th July, 1892).—The skull was penetrated over the frontal bone by an irregular aperture, the site of the compound fracture and the trephine openings. There was purulent lepto-meningitis extending from this aperture on the frontal vertex all over the right side of the brain, and following the course of the great anastomotic vein to the base. The left side of the vertex was affected with serous lepto-meningitis, as were likewise the posterior portions of both hemispheres. There was marked suppurative lepto-meningitis extending from the optic chiasma backward to the cerebellar fossa. In the latter there was a considerable collection of pus on the anterior aspect of the hemispheres.

The frontal lobe of the right hemisphere was in a state of acute encephalitis, partly red softening and partly greyish œdematous masses, the seat of the former abscess. There was little pus remaining in the frontal lobe. The brain tissue at other parts was congested but healthy. Both ventricles contained fluid—in the left simply turbid, on the right semi-purulent serum. The right choroid plexus was covered with thick pus. There was pus in the third and fourth ventricles. The whole of the spinal cord under the theca was covered with a fluid layer of pus, which welled out on the dural covering being incised. A portion of the cord enclosed in its theca was removed for histological examination.

CASE XVIII. *Cerebro-spinal meningitis and extradural abscess in cerebral and cerebellar fossæ with partial thrombosis of sigmoid sinus. Coming under observation on seventh day of her illness. Duration of illness twenty-three days. Recovery.*

M. T., aged 24 years, housewife, admitted to Royal Infirmary on 5th October, 1891, suffering from symptoms of cerebro-spinal meningitis at an early stage, arising from chronic otitis media.

History.—*Heredity.*—Her mother, and a brother aged five years, were dead, the former dying from consumption, the latter from "inflammation of brain." Father well. *Personal.*—Had been in good health until twelve years of age, when she had measles, after which she had a purulent discharge from the left ear, which continued for some months, and then ceased. During the twelve ensuing years the discharge from the ear recurred very frequently, sometimes when she had "catarrh," and occasionally without any apparent cause. The discharge was very fœtid.

Present Illness.—A week ago she was exposed to cold on a wet night, after which in the left ear she had pain, which in a few hours extended all over the back of neck and head. On the third day of her illness she had a severe rigor, the pain had spread down her spine, and she had shooting girdle pains round upper part of trunk. Vomiting was frequent, giddiness pronounced whenever she attempted to raise her head from the pillows, she had marked photophobia, and was semi-delirious.

Condition on Admission.—On the seventh day of her illness, when she entered the Infirmary, she was semi-delirious, lay with her head

retracted, the muscles of the neck and back of trunk were stiff, as were likewise the masseters. The limbs were exceedingly feeble, the vomiting was incessant. She had frequent yawning—the mechanical opening and shutting of the mouth variety. The pain in back of neck and in head still evidently troubled her, as she put her hand over these parts and moaned. Pressure over the left internal jugular at its upper third caused pain, as did likewise pressure in the apex of the posterior cervical triangle. There was likewise a little tumescence over these parts. It was difficult to ascertain the state of her sensations owing to her perturbed mental state.

Patellar reflex was exaggerated, and ankle clonus was present, and the induction of the reflexes occasioned pain.

Ocular Examination.—Except for photophobia, the sight seemed good as far as could be properly tested. No ocular paralysis; pupils equal, medium, responded to light and accommodation.

Ophthalmoscopic Examination.—In right eye congestion of retinal vessels with roughened outline of veins, opacity of nerve fibres as they passed over the margin of the disc. Traces of the congenital cup with venous pulsations. In left eye the outline of the nerve was rather veiled, with slight indications of opacity of nerve fibres as they passed over the margin of disc.

Ears.—Right, normal, functionally and otherwise. Left, membrane ulcerated and granulation tissue projecting from middle ear, foul-smelling discharge not profuse.

Taste and smell natural.

Temperature was variable from 100° F. to 103° F., pulse over 100.

Operation (7th October, 1891).—The mastoid antrum was filled with granulation tissue and foul-smelling pus. The tegmen tympani was eroded, a little pus existing between the skull and the dura. The tympanum was likewise filled with granulation tissue, and the ossicles—malleus and incus—were imbedded in adherent granulations. These were removed, both bones showing evidence of erosion. The mastoid erosion passed backwards towards the sigmoid groove, the bone at this point being eroded and dark-coloured. The sigmoid sinus was exposed for about half an inch. It was covered with granulation tissue and pus, the purulent secretion being extradural. The sinus was somewhat thickened, but was not absolutely thrombosed. The whole was washed out, the granulation tissue removed, and the cavity filled with iodoform and naphthaline.

Patient improved quickly after the first week, and made an uninterrupted recovery. The aperture in the mastoid was "papered" with epithelium, and the middle ear communicated with the skin by an aperture behind the ear.

June 10th, 1892.—Patient is now very robust, feels stronger than she has ever been, and is quite fit for her household duties. There is now no discharge from ear. Her hearing in left ear is defective ; the watch can be heard, however, about three feet distance, and ordinary conversation can be made out with some difficulty. She hears better when the aperture behind the ear is not plugged with cotton wool.

The right fundus is normal ; the left has the outline of disc still hazy.

Purulent encephalitis.—In the formation of purulent encephalitis, besides the swelling of the cerebral substance, there is extensive serous and leucocytal exudation and extravasation of red blood corpuscles, which is a very frequent occurrence in acute inflammations of the brain.

Red softening.—When many of these minute hæmorrhages coalesce they give rise to the condition known as red softening.

The red corpuscles are effused into the perivascular spaces or into the brain tissue itself, and are soon followed by leucocytes, which are exuded in large numbers from the vessels. The affected area of the brain is œdematous and swollen, and on section projects above the level of the healthy structures. The pressure thus occasioned may *aid* in producing the pain experienced by the patient at the very outset of the encephalitis, especially when the ganglion of the fifth and its recurrent meningeal branches are included in the area of pressure. As the exudation continues the nerve tissue degenerates, a process of molecular disintegration ensues, in which the minute particles of brain tissue become mixed with the disintegrating leucocytes which have formed into pus cells.

CASE XIX. *Meningo-encephalitis in process of abscess formation, involving left motor area and temporo-sphenoidal lobe, probably arising from marasmic thrombosis of longitudinal sinus. Patient in state of exhaustion. Trephining to relieve pressure symptoms, formation of false hernia cerebri at moment of opening the skull. Slight temporary improvement. Death from exhaustion on tenth day after operation. Admitted on sixtieth day of illness. Duration of disease about seventy days.*

The following is an interesting though incomplete case in which a false hernia cerebri formed at the moment of opening the skull.

M. A., a girl aged 12 years, was sent by Dr. Frew of Kilmarnock into Royal Infirmary in 1884 with symptoms of advanced meningo-encephalitis, involving a considerable area of the left side of the cerebrum.

History.—*Heredity.*—No constitutional tendency to tubercle or syphilis. *Personal.*—She was a weak girl, though previously healthy.

Present Illness.—The attack commenced two months prior to admission with shiverings and vomiting, followed by double vision, and by delirium alternating with periods of stupor.

During the first week there were spasmodic twitchings of the muscles of the limbs, especially those of the right side, the head being drawn back, considerable rigidity of the muscles of the neck being present. During the

FIG. 43.—ENCYSTED CEREBELLAR ABSCESS.

Aperture left by cerebellar abscess which had become encysted.

FIG. 44.—SECTION OF ENCYSTED CEREBELLAR ABSCESS.

The cerebellum, showing section of encysted abscess in its right lobe.

To face page 102.

first fortnight she complained of loud ringing noises in her left ear, and, as this subsided, excessive pain over the left side of the head was experienced. She recovered somewhat during the fourth week, and remained slightly relieved until the seventh week, when a new series of symptoms developed. The pain on the left side of the head became greatly aggravated, followed by severe clonic spasms of right side of face, right arm, and right leg, these parts becoming paralysed after the convulsion had passed off. Forty-eight hours after this attack, the loud noises in her ears recurred, accompanied by a sensation of dizziness, the objects in the room appearing to her as if they rose up and moved round her, while she experienced the sensation of falling. This was succeeded by excruciating pain in the head, causing her to give vent to loud piercing shrieks, which continued for hours, with short intervals, during which she lay exhausted and moaned. Towards the evening of that day she had two convulsions, which began on the right side of face, extending rapidly to the right arm and right leg. After each of these she lay for a couple of hours in a state of insensibility. When she recovered sensibility, the pain in the head had greatly diminished, but she had right-sided hemiplegia.

State on Admission.—On entering the Infirmary her condition was as follows:—She was a pale, emaciated girl, who lay with her head thrown back, the muscles of the neck being rigid. The veins in the front of the head were greatly distended. For the most part she was semi-conscious, giving monosyllabic answers to questions, but occasionally became fairly intelligent and spoke about her feelings with tolerably distinct articulation. There was right hemiplegia, the tongue being protruded toward the right. Taste and smell were unimpaired. Hearing on the left side was defective, there was no discharge from the left ear, and the membrana tympani was normal. There was slight squint, diplopia when both eyes were open, the false image being on the right side. The pupil of the right side was somewhat larger than that on the left, both reacting sluggishly to light. Ophthalmoscopically there was congestion of the retinal vessels, though no distinct optic neuritis. She complained of pain over the left side of the head, and on percussion the pain became acute over the posterior parts of the parietal and temporal bones. Besides the above, she occasionally complained of vertigo, "as if the tables and chairs were moving round her head," and had the sensation while she lay flat in bed, "as if she were falling." Her temperature was 101° F. to 102° F. There was a tremor over the left side when she attempted movement.

The day after admission she became delirious, with occasional lucid intervals. Towards evening coma became complete, with dilation of both pupils, lividity of the face, stertorous breathing, slow pulse, and subnormal temperature (97° F.).

With regard to the diagnosis, it appeared from the fact that there had been right-sided convulsions, followed by right motor hemiplegia with preservation of sensation, that there was a lesion of the superficial parts of the whole left motor cortex. The swollen condition of the veins in front of the head, brow, and face were indicative of intracranial venous obstruc-

tion, possibly a marasmic thrombosis of the longitudinal sinus. If this had occurred, the thrombic process probably originated in the left cerebral veins leading to the sinus, and red cerebral softening of the motor area might thus have resulted. The long illness, and the convulsions coming on in the seventh week, lent countenance to this view. On the other hand, the defective hearing in the left ear, the loud ringing noises and the vertigo while in bed, rather indicated involvement of the internal ear on the left side. (Subsequent experience has shown that extensive disease of the middle and internal ear is occasionally present, though there are no signs of it externally, the membrana tympani being intact.) It was probable that there was a meningo-encephalitis passing on to the formation of abscess, if pus had not already formed. A fatal termination was imminent. It was questionable whether surgical interference could cure, though it might afford temporary relief. Pressure symptoms somewhat suddenly supervened and became very pronounced. It was decided to relieve them.

Operation.—Trephining was performed towards the lower and posterior border of the motor cortex.

The disc of bone being elevated its inner surface was seen to be thinned and eroded, having a worm-eaten appearance, and on examining with the probe, the inner surface of the skull for a considerable distance round the trephine opening was found to be similarily affected. The dura mater was covered by a layer of plastic effusion, the external surface of which was indented corresponding to the irregularly eroded surface of the bone. The dura mater was much thicker and more tense than usual. When incised along with the adhering inner membranes, a dense reddish yellow mass was quickly extruded, so that within a few seconds from the opening of the membranes a large false hernia of the brain was formed and lay on the exterior of the skull. The first portions extruded contained a little pure pus, which imparted a yellowish colour to the mass; the subsequent portions seemed to be made up in great part of capillary hæmorrhagic extravasations, such as are found in red softening (see microscopical examination). This somewhat spherical protrusion was at first about two inches in diameter at its base, and it protruded beyond the level of the skull for nearly one inch and a quarter The size of this protrusion and the rapidity of its formation were such as would have caused considerable apprehension had it not been remembered that such protrusions do not consist of cerebral substance alone, but are largely made up of exudation cells and minute hæmorrhagic extravasations. In order to facilitate drainage a second disc of smaller size was subsequently removed from the squamous portion of the temporal, exposing the temporo-sphenoidal lobe. The same phenomena here presented themselves. The membranes were, however, more affected, being very pulpy and converted in great measure into granulation tissue. A thin layer of pus lay on the surface of the cerebral softening, which protruded to a slight extent beyond the level of the skull, the pressure having been greatly relieved by the first opening.

Course after Operation.—During the first twelve hours the false hernia cerebri increased in size, nearly doubling the dimensions it had assumed

at the end of the operation, but at this point it remained stationary, maintaining the same proportions till the end. Though it was apparent that recovery could not take place, yet the patient seemed to be relieved after the operation, and, though excessively weak, she somewhat improved during the first seven days, and then gradually sank and died from exhaustion on the tenth day after the operation and about the seventieth of her disease.

Unfortunately no post-mortem was permitted. The false hernia cerebri fell considerably after death. During the progress of the case, however, portions of it were repeatedly examined microscopically, and diligent search was made for evidence of the presence of brain tissue, but none was found. The mass was made up principally of red blood corpuscles in various stages of degeneration, thickly interspersed with leucocytes. In some parts pus corpuscles were found in groups, forming abscesses varying from the size of a barley grain to that of a horse bean.

White softening.—There may be, with very little hæmorrhagic extravasation, a primary leucocytal exudation which may directly pass into pus production and molecular disintegration as above.

When this disintegration occurs the softening process considerably relieves the pressure, and the pain formerly experienced by the patient greatly diminishes.

When a pathogenic embolism occurs in a cerebral artery or vein situated in the white substance of the brain, hæmorrhagic extravasation ensues, accompanied or shortly followed by an exudation of leucocytes, which infiltrate the extravasation and the brain tissue in the vicinity. These leucocytes rapidly degenerate into pus, while the neighbouring nerve tissue disintegrates and liquefies. These emboli may be accompanied by anæmic or hæmorrhagic necrosis, according as an artery or a vein has been blocked. This may be indicated by the appearance of the parts during operation: when they are œdematous and glistening they are anæmic, and therefore due to arterial thrombosis, and when they appear as reddish-brown sloughs they are hæmorrhagic and due to venous thrombosis. When large arteries are blocked, necroses of extensive areas may occur which may be afterwards separated. Such large necrotic portions may also arise from very acute and infective inflammation of the brain (see Case XX).

Character of pus found in cerebral abscess.—The pus which the abscess may contain varies much in colour and consistence. When mingled with much disintegrated tissue which has been infiltrated with extravasations of blood, it is of a dark brown

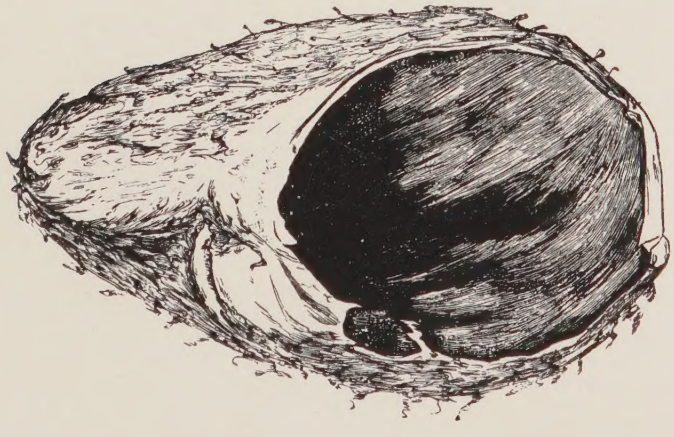
colour. When free from such extravasations, it assumes a greenish yellow tint, probably from the presence of the bacillus pyocyaneus. When very fœtid it is thin, serous, and generally contains many minute sloughs. The fœtor is usually identical with that perceived from the pus in the middle ear, and the same bacillus—the bacillus pyogenes fœtidus—has been found among others in the pus from the middle ear, and from the abscess cavity. Micrococci are abundant in these abscesses, especially when they are opened at a somewhat early period, and not the less so when the abscess has but little fœtor. Streptococci and staphylococci have almost always been present in those abscesses the pus of which has been taken from the cases under the author's care. (On this point see further under section of Micro-organisms the Cause of Intracranial Pyogenic Processes.)

The tissue surrounding and forming the boundary of the acute abscess is actively inflamed. The vessels may be seen in all stages of inflammation, from hyperæmia in the outer zone to thrombosis in the inner. Surrounding the vessels are masses of exudation cells mingled with extravasated red blood corpuscles, and the glimpses of brain tissue which may be had in the less inflamed parts show it to be softened and disintegrating. When the inflammation has been great the vessels are scarcely recognisable on account of the exudation cells not only filling the surrounding parts, but also the vessel wall, and in great measure occupying its interior, the red blood corpuscles being faintly seen in some, while in others they have entirely disappeared. Irregularly distributed over the surface of the abscess are portions of minute sloughs of an œdematous greyish appearance, with pus in their periphery and in the pockets or the sinuosities in the abscess wall. The surface toward the abscess is flocculent, shaggy, and irregular in outline. Occasionally little naked thrombosed vessels project from its surface into the abscess cavity at parts where the molecular disintegration has proceeded more actively in the soft brain tissue surrounding the vessel. As in processes of ulceration in other parts of the body, the small vessels become thrombosed in advance of the molecular necrosis, and so hæmorrhage into the abscess is prevented. Occasionally, however, the process of disintegration is more rapid than that of thrombosis, and bleeding occurs into the abscess cavity, giving rise to symptoms of apoplexy. The zone of brain tissue surrounding the abscess is œdematous, and

FIG. 45.—ENCAPSULATED ABSCESS.

External view of capsule of abscess taken from Case XXX. A portion of wall of abscess has been removed. The external surface is shaggy and flocculent.

FIG. 46.—ENCAPSULATED ABSCESS.



Internal view of the capsule of abscess taken from Case XXX. The internal surface of capsule is smooth and glistening.

To face page 106.

its vessels are hyperæmic. There is thus in the periphery of an acute abscess no other wall than a softened and disintegrated cerebral tissue filled with exudation cells and extravasated blood.

The pia mater and arachnoid tissue over an area of cerebral degeneration have often a milky and turbid appearance.

The encephalitis surrounding the abscess extends to a distance beyond the abscess itself. Were this zone of encephalitis to embrace a part of the brain whose function was known and capable of external manifestation, symptoms might arise which might lead one to localize the abscess at a distance from its actual seat.

Encapsulation of the abscess.—When the process of molecular disintegration which has produced the abscess ceases, and the brain tissue possesses sufficient vitality to assume a formative action, the debris of disintegrated tissue, granular cells, leucocytes, blood, and inspissated pus become entangled in the meshes of fibrin, the elements for the formation of which are poured out from the living tissue. Into this mass large numbers of leucocytes penetrate. Some of these form into elongated spindle cells, bundles of which may be seen interspersed throughout the membrane. At an early period it contains no bloodvessels, and, therefore, secretion from its surface must be limited to the few leucocytes which have travelled through it from the living tissue. Later a few bloodvessels may project from the living tissue into the membrane at a point where it is abundantly penetrated by leucocytes. These vessels are, however, of the most primitive description, such as may be seen in imperfectly developed granulation tissue. They are at first so small and imperfectly developed that they can only supply nutriment to the leucocytes in their immediate neighbourhood. As they are generally to be seen on the side of the encapsulating membrane next the living tissue, they cannot provide many exudation cells for fresh pus formation.

The pus in the abscess thus becomes encapsulated by a layer of membrane of very low vitality which shuts off the living brain tissue from the dead pus within. When this capsule is complete on all sides the abscess becomes to a great extent stationary First, because the process of molecular disintegration has ceased Secondly, as the leucocytes which penetrate for some way into the living wall of the abscess, and which might be converted into pus were they shed on the inside of the capsule, are principally caught

by the meshwork of fibrin, so that few pass into the abscess cavity; and as the lining membrane increases in thickness the difficulties of transmigration of the leucocytes increase, both on account of the greater distance they have to travel, and the increasing formation of fibrous tissue bundles, which are interspersed throughout it. Thirdly, this same membrane, although it does permit the passage of serum from the pus cavity to the vessels in the living tissue, yet prevents the absorption of the particulate portions of the disintegrated pus. The thickness of the abscess capsule varies considerably, from one to five or more millimetres.[1] It is generally smooth internally, and rather ragged and flocculent externally—next to the living brain tissue. It is for the most part rounded or ovoid, while the boundary of the acute abscess is generally irregular in outline. The time required for the formation of the abscess capsule is indefinite, and depends on the condition of the part and the character of the inflammation. Some abscesses of at least three weeks' duration were found to be without capsules.

Description of encapsuled abscess taken from case.—In Case XXX., when the peripheral abscess was opened, the primary abscess, contained in its cyst-like sac, was seen floating in a pool of pus. On the capsule of this abscess being examined, after long preservation in spirit, it still retained its original oval shape, though it had shrunk considerably in size. It then measured two and a half inches in length and one and a half in its short diameter (62 by 37½ mm.). Its walls were of variable thickness, from two to five millimetres. Before prolonged soaking in spirits they were much thicker. Its exterior was rough and flocculent, probably corresponding to the indentation of the living tissue, with which it had been in contact prior to the pus formation in its periphery. (See Fig. 45.) Its interior was smooth, glistening, and had a polished aspect. (See Fig. 46.) The wall on transverse section, though apparently homogeneous, could be split into two or more layers, especially an inner one, about one millimetre in thickness, which could be separated from the outer. A portion of the wall was removed for histological examination. To the naked eye it appeared to be of a soft pulpy nature; it easily crumbled, and imparted a somewhat unctuous sensation to the fingers. On microscopical examination, it was seen to consist of masses of round cells, a few blood channels of

[1] See description and dimensions under case.

the most rudimentary granulation type, confined to the layer of tissue nearest the brain, and here and there interspersed throughout the outermost layers, spindle cells in bundles. There was no distinct layer of connective tissue.

The contents of a chronic cerebral abscess are said to be odourless, of a greenish colour and of acid reaction. The contents of this encapsuled abscess were examined shortly after the removal of the cyst from the brain. They were very fluid, of a pale straw colour, and with a heavy but not very fœtid odour, though the fluid in the peripheral abscess was highly offensive. Four hours after having been poured into a narrow glass vessel, the contents had separated into two portions. The upper two-thirds consisted of a cloudy serous fluid, the remainder was an opaque mass of a muddy yellow colour. The latter consisted of disintegrated corpuscular elements, minute oil globules, and some swollen fatty pus cells. Occasionally, there is formed in the periphery of those encapsulated cerebral abscesses, a firm layer of connective tissue, such as might form round any foreign body, which remained physically and chemically quiescent. When such a barrier is formed, the living brain tissue is well protected, especially as the connective tissue becomes more contracted and avascular the longer it exists. The capsule within the connective tissue is occasionally found calcified, and on one occasion even the inspissated purulent contents participated in the calcification (*Ed. Med. Journal*, Fenman, Oct., 1879). The reference quoted is, however, open to doubt—the chalky mass may have been formed in pathological deposits other than pus.

Cerebral abscess may become absorbed.—This wall may not, however, remain permanently in the condition described. Changes take place in it from the side of the living tissue. The circumferential pressure of the brain upon the abscess may facilitate the absorption of the fluid portion of the pus. Well-formed vessels may be thrown out from the living brain tissue, and penetrating the capsule, may thus aid in the absorption of pus debris through phagocytic action. In this way it is possible for a considerable portion of a small cerebral abscess to become absorbed.

Circumstances under which an encapsuled abscess may increase from within.—On the other hand, if the capsule be replaced in part by a layer of vigorous granulation tissue, a fresh supply of pus may be formed which may augment the size of the abscess and exercise pressure from within upon the capsule which is thus

apt to become thinned at parts. The abscess may then burst either into the ventricles or into the subdural space. In either case very serious symptoms ensue.

Cerebral abscesses may empty themselves externally by aid of pathological processes alone.—In some instances, the encapsulated abscess in the temporo-sphenoidal lobe, lying over a perforated tegmen tympani, to which the brain and its membranes have become soldered by inflammatory action may by its weight cause thinning of the lower part of its capsule, and the absorption of the layer of brain tissue intervening between the tegmen tympani and the capsule of the abscess. The obliteration of the interspace between the dura and the pia mater may occur from within outwards, as well as from the opposite direction, the molecular disintegration occurring slowly and affording time for the advancing plastic effusion to effect the soldering of the brain membranes. Under such circumstances, the pus may gradually find its way into the middle ear, and so escape externally.

By a somewhat similar process, the pus from an abscess in the frontal lobe has found an exit through the perforated cribriform plate of the ethmoid into the nose. Cases are recorded in which the clinical history indicated cerebral abscesses which discharged in this way, the patients afterwards recovering. In an instance which came under the author's observation, an abscess in the cerebellar fossa which had penetrated the membranes escaped by way of the posterior condylar foramen—the contained vein being in a state of disintegrating thrombosis—and formed an abscess in the upper portion of the posterior cervical triangle, beneath the deep fascia, the pus within the skull and that without being continuous through the foramen.

An *otorrhœa cerebralis*,—provided one understands by that term leakage by pathological processes from an abscess in the brain, through a pre-existing osseous erosion by which the infective matter originating the abscess entered the cerebral tissue,—may actually become established. Gruber, however, relates a case in which the cerebral abscess emptied itself into the middle ear, which, in his opinion, was previously healthy.[1] No such case has ever been seen by the author.

[1] Otitis cerebralis—Gruber, *Text-Book of Diseases of the Ear*. London, 1890. "In rare cases, purulent median otitis has been caused by a suppurative inflammation of the structures in the cavity of the cranium. . . . The author has been able in certain cases to diagnose the condition accurately. The last case occurring in his hospital practice was also seen

Cerebral abscesses may burst or leak into the subdural tissue or into the ventricles.—Were the cerebral abscess to burst into the subdural space before the latter was fixed by adhesions, acute suppurative lepto-meningitis would ensue, which, if not promptly relieved, would extend over the whole cerebro-spinal system, and in all probability end in death. Instead of penetrating to the surface of the brain, the pus may gravitate toward, or the molecular disintegration may proceed in the direction of the lateral ventricle, the descending horn of which is in immediate vicinity of abscess situated in the temporo-sphenoidal lobe. If there be a slight leakage of the pus into the ventricle, or even if the pathogenic micro-organisms contained in the abscess find their way into the ventricular fluid, intense irritation ensues, though if the transmission of the peccant matter be slow, the inflammation may assume at first a subacute course. The abscess may however burst suddenly into the ventricles, when a violent suppurative lepto-meningitis is quickly developed, exhibiting a train of serious symptoms often ending fatally, within a very short period. (See Case XXXIII.) Cerebellar abscess may likewise burst into the membranes, and set up cerebro-spinal meningitis.

Can an abscess form in the brain from infective otitis media purulenta and remain in situ *while the middle ear disease becomes cured?*—Such a condition is highly improbable, though a case is given by Gruber of Vienna, witnessed by himself, which showed at least that healing had taken place in the external parts of the ear while the abscess existed in the brain. Dr. Gruber's patient was a labourer, aged forty years, who had right purulent otitis media with perforation of the membrane, followed by mastoid periostitis and abscess, which was cured at the end of six weeks. Three months afterwards he returned complaining of severe headache but "having no fever or other apparently important symptom." He was, however, admitted to the hospital, after which he had high fever, and on the third day he became insensible and died on the fifth day. On post-mortem examination being made an abscess the size of a hen's egg was found in the right temporal lobe, its walls giving evidence that it was of considerable duration. In the corresponding temporal bone no abnor-

by Professor V. Shrötter. In this case, the pus derived from a cerebral abscess discharged itself through, until then, a healthy ear, and its origin could be recognized at a considerable distance by its characteristic odour. Berndgen relates a similar case which he observed.—*Monatschrift für Ohrenheilkunde*, Bd. xi."

mality could be seen beyond a moderate thickening of the drum membrane and of the lining membrane of the mastoid cells. There can be little doubt, however, that the origin of the abscess is to be referred to the middle ear disease from which recovery had taken place.[1]

A fresh abscess may form in the periphery of the encapsulated one.—The tissues in the vicinity of the encapsulated abscess are not always tolerant of the foreign body in their midst. They suffer from an amount of compression, which is apt to induce degeneration and atrophy. Slight causes may then occasion in them fresh œdema and inflammation, just as encephalitis occurs round a tumour. If this encephalitis becomes purulent, a new abscess may form in the periphery of the old one, leaving the latter intact within its capsule. It is probable, however, that a leakage of the contents of the abscess into the compressed tissues in the vicinity may set free from imprisonment inflammatory elements (micro-organisms) which, meeting with fresh pabulum, regain their former vitality, and start afresh the inflammation in the tissues on the outside of the capsule. In such a case, an extramural abscess develops in the circumference of the old one, which it may set so completely free from its attachments that the encapsulated abscess may be found floating in the pus of the peripheral or secondary abscess. This was observed in one of the author's cases, in which the primary abscess contained in its cyst-like sac was seen floating in a pool of pus. (See Case XXX.)

The size of the cerebral abscess cannot be estimated by the degree of pressure produced by the abscess.—The size of a cerebral abscess cannot always be estimated by the degree of pressure produced. It is not like a solid tumour, which, to the extent of its bulk, pushes aside the living tissue in its vicinity. The molecular disintegration by which the abscess is formed hollows out an aperture in the brain which is filled by the molecular debris and pus. The abscess will, therefore, not produce the same amount of pressure on the adjacent structures as a solid tumour of the same size, so that pressure symptoms occasioned by an abscess would be less than would be produced by a tumour of corresponding bulk. On the other hand, the symptoms due to œdema and inflammatory action would probably be greater in the acute stage of abscess formation than in most tumours. The pressure exerted by the

[1] English Translation of Gruber's *Diseases of the Ear*, p. 381.

abscess on the adjacent structures will depend in part upon whether the periphery of the abscess is sufficiently inspissated and resistant to convey force from within. The pressure is moreover not wholly occasioned by the size of the abscess, but also by the amount of inflammatory exudation, and consequent swelling of the peripheral tissues. When the abscess is of considerable size, the convolutions in its vicinity are flattened and their substance is anæmic. The pressure of a large abscess in the temporo-sphenoidal lobe may produce effects upon the motor area of the cortex, commencing in the face centre and, hours or days subsequently, affecting the arm with paresis. As the brachial paresis becomes pronounced the facial paralysis due to the cortical lesion becomes more marked, or, perhaps, absolute, showing that the arm centre has been influenced through the facial, *i.e.* from below upwards. The articulating and speech centres may be affected secondarily, according to the situation of the abscess in the temporo-sphenoidal lobe. Paralysis of the third nerve, which, when there is a large abscess, often accompanies that of the motor cortical centres, may also arise from communicated pressure,[1] and when present along with the other motor phenomena described above, it indicates a large abscess or pressure of some kind, extending from the temporo-sphenoidal lobe.

Pressure on the middle lobe of the cerebellum, whether directly from an abscess in that lobe or from pressure exerted through the cerebellar hemispheres, gives rise to many of the symptoms found with tumours similarly situated.

Size of cerebral and cerebellar abscess.—The size of a cerebral abscess varies very much—its cavity containing from a few drops of pus to five or more ounces—and depends somewhat upon the locality in which it is situated. If located in a very vital part, life may be destroyed at a very early period in its formation, and before the abscess can attain any large size. If, on the other hand, it be seated in and confined to the temporo-sphenoidal lobe, a very large portion of this lobe may be destroyed before the patient succumbs; hence abscesses containing three and four ounces of pus are not infrequently found here. In one fatal case, the whole temporo-sphenoidal lobe had disappeared, its place being taken by pus which filled the greater part of one half of the middle cerebral fossa. Cerebellar abscesses are usually small, though when they have burst into the cerebellar fossa, they

[1] See Atlas of Head Sections, Plates 42 and 45.

may contain four ounces of pus. In such cases, it is probable that the pus from the abscess, though found between the membranes, may yet have been circumscribed by pathological limitations, otherwise the whole spinal canal would be full of pus Once an abscess has burst into the subdural space, it may extend into the arachnoid cisterns and spinal canal, inducing acute suppurative cerebro-spinal lepto-meningitis.

Hæmorrhage from large brain vessels rarely takes place into the cavity of an abscess, but when it does it produces sudden and serious symtoms, resembling those of ordinary apoplexy.

Multiple abscesses from ear disease or from infective traumatism are very rare, unless those conditions have induced general pyæmia. It is said that four-fifths of the multiple abscesses of the brain are pyæmic. Case No. XXXVII. is an instance of multiple cerebral and cerebellar abscess arising from ear disease.

Lowering of temperature and slowing of pulse rate.—The temperature is as a rule lowered in abscess of the brain, but the cause of this depression is not clearly ascertained. In some forms of idiopathic encephalitis, where the membranes are free from involvement, the temperature is likewise about or slightly below the normal. A similar condition of temperature exists in some low forms of infective peritonitis, and is then generally due to a form of toxæmia. In abscess of the brain the low temperature is accompanied by slow pulse, which is often a marked feature. Whether the lowering of the temperature be due to pressure on the brain is questionable, as the increase in the temperature which follows evacuation of the contents of the abscess might be attributed to other causes, such as the removal of the infective organisms, which through the blood might have had some action upon the heat centres. Against the latter hypothesis, there is the fact that when an abscess of the brain which has induced a low temperature afterwards leaks into the membranes or into the ventricles, presumably the same organisms which the abscess contained set up a very high temperature, increasing rapidly from subnormal, at the rate of at least a degree per hour to 104°-105° F.

There can be no doubt that the pulse is slowed in cerebral abscess owing to the pressure exerted, as after the abscess has attained a certain size its rate decreases as the abscess

FIG. 47.—NECROSIS OF PORTION OF BRAIN.

Extensive necrosis of left temporo-sphenoidal lobe. Photographed at post-mortem immediately on removal (Case XX.).

To face page 114.

FIG 47.

increases, and the moment the pressure is relieved by evacuation of the pus there is a sudden bound in the pulse rate, from for instance 40 to 120 per minute. At the latter rate it remains for some minutes, when it gradually slows down to perhaps 90 or even 80 per minute, at which rate it remains for days. That this diminution in the pulse rate is due to pressure, and not to the presence of pus in the brain, may be inferred from the fact that intracranial hæmorrhage, when excessive and localized, produces a like result, and the previously slow and subnormal pulse is at once relieved when the blood is removed from the interior of the skull or its brain membranes. In certain stages of tubercular meningitis attended by serous effusion, either meningeal or intraventricular, the pulse rate is slowed when pressure is exerted on the brain by the increase of the serous effusion, and is instantly augmented in rate when the serous fluid is drawn off. The same results in many cases of tumour exerting pressure on the brain, and also from acute encephalitis occasioning swelling of the affected part. The point referred to above must here be remembered; the pus in the cerebral abscess *may* only occupy the area destroyed by the molecular disintegration, and, if so, little pressure can be exerted on the brain, and therefore little alteration occurs in the pulse rate.

The temperature, however, also rises after the evacuation of the pus, but much more slowly, as one would expect, from the difference in the mechanisms regulating the temperature, and the pulse. In one instance, within an hour of the relief of an abscess in the temporo-sphenoidal lobe, the temperature had risen by a degree. After evacuation of the abscess the temperature generally does rise above normal, and in a few days thereafter returns to its normal state.

Necrosis of a portion of cerebral tissue may arise in several ways. Infective embolism or thrombosis of the main vessel supplying a portion of the brain leads to anæmic gangrene of the part. Embolism of the main veins is apt to induce gangrene from the intense pressure set up in the brain by the extravasation due to the blocking of the exits of the blood-stream—hæmorrhagic necrosis. Occasionally, also, the localized pressure of the part engorged with extravasation may exert influences upon the neighbouring brain tissue, its vessels and its membranes, leading in these to disorganization and death. The intensity of the inflammatory

action, depending on the nature of the micro-organisms which are the exciting cause, may also induce rapid advancing necrosis. The membranes overlying the part may likewise participate in the local death, though this is seldom found. Frequently mingling with the pus in brain abscess, there are sloughs varying from the size of a pea to that of a filbert. Usually these are greyish-white, œdematous masses, but when there has been much cerebral extravasation they may assume a dark brown colour. For the removal of sloughs of this kind, a larger opening is required in the brain than would be necessary for the mere exit of the pus. If these sloughs be not removed, the pus will in all probability reaccumulate and require re-evacuation a second or a third time, until the slough either has become particulate and discharged, or has ultimately become absorbed.

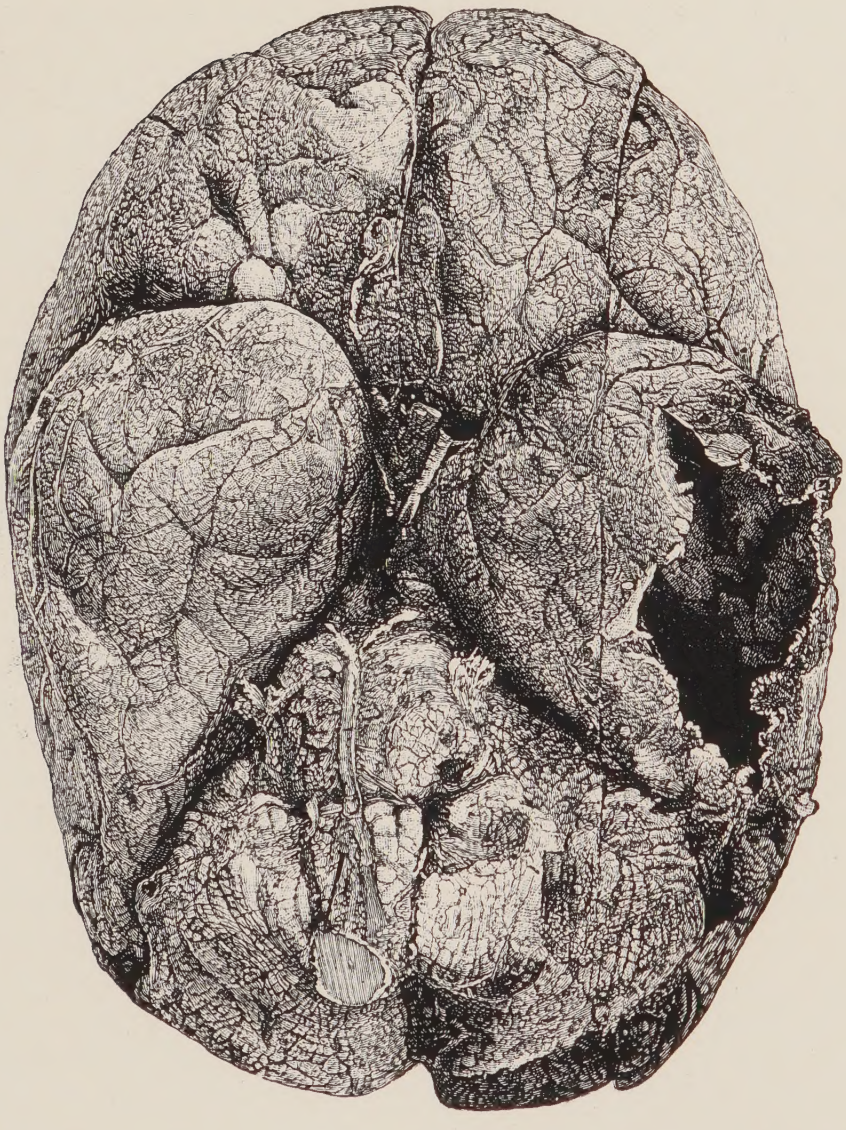
In one instance, necrosis of a large portion of the temporo-sphenoidal lobe, along with its overlying membranes, including the dura mater, was found as a sequela of infective otitis media. The dura was like wash leather in appearance and to the touch. The brain was at the corresponding part sodden and greyish white and œdematous, at others of an intense dark brown colour. There was a little pus surrounding the slough and between the membranes. (See Case No. XX.) Though it is possible that this necrosis arose from the intense pressure exerted upon the membranes and brain by the extravasated blood, and leucocytal exudation, the vessels at the base of the temporo-sphenoidal lobe becoming occluded from the pressure within the brain, it is more probable that it arose from infective thrombosis.

CASE XX. *Necrosis of the temporo-sphenoidal lobe with infective thrombosis of sigmoid sinus, and slight involvement of longitudinal sinus, with pneumonia and pulmonary infarctions. Death. Admitted on twenty-first day of illness. Duration of illness twenty-nine days.*

D. C., aged 32, forgeman, admitted to Royal Infirmary, 21st May, 1891, affected with extensive necrosis of the left temporo-sphenoidal lobe, involving the wall of descending horn of left lateral ventricle; thrombosis of the sigmoid sinus and internal jugular vein, and infective pulmonary infarction, arising from purulent otitis media of twenty-eight years' duration.

History.—*Heredity.*—Good. *Personal.*—When four years old, patient had an otorrhœa following measles; when fourteen years old, he had "polypi" extracted from the external ear, and further growths were removed therefrom

FIG. 48.—NECROSIS OF BRAIN.



Aspect of brain after hardening in spirit, showing cavity in temporo-sphenoidal lobe after removal of sloughs (Case XX.).

To face page 116.

three years later. His habits were intemperate. Seven years ago he had inflammation of the kidneys, and on two occasions—the precise dates not known—he had inflammation of the left lung.

Present Illness.—Three weeks prior to admission, he was attacked by pain commencing in the left ear, and radiating all over the left side of head. On the same day he had a severe rigor. A week afterwards he had another shivering, and two days subsequently had a difficulty in speech, which at a later date was diagnosed as aphasia by Dr. Finlayson, who saw him in consultation with Dr. Barr.

Condition on Admission.—On the 21st May, 1891, when admitted to the Royal Infirmary, his condition was as follows :—

His face had a rather dusky, earthy, look ; and over the right side of it there was a slight paresis of a central origin. The right arm and leg were more feeble than the left. He moved the left more freely and more frequently than the right, although he was a right-handed man. The dynamometer registered about 15 on the left, while the grasp of the right was not perceptible. There was a difficulty in being sure about this observation owing to patient's mental obscuration.

There was only slight paresis of the left ocular muscles, with an occasional squint. The conjunctiva of the right eye was somewhat injected, but apparently he had previously had an injury to that eye. The pupils were unequal, left very slightly larger, though both were small. There was considerable dilation of the vessels of the fundi, but there was no actual optic neuritis. The ocular examination was rendered difficult owing to the constant movement of his eyeballs.

Ears.—There was an escape of foul-smelling pus from the left ear, its membrana tympani had disappeared, and granulation tissue projected from the tympanum. The right ear was normal. Hearing power could not be determined owing to patient's mental condition. There was pain on pressure over the upper two-thirds of the internal jugular, and in upper third of posterior cervical triangle.

The knee jerks were slightly increased, and there was slight ankle clonus. The cutaneous sensibility could not be determined.

Mental Condition.—There was considerable mental obscuration, a want of comprehension of simple questions, and a certain sluggishness of cerebration, coupled with restlessness, nervous agitation, and frequent erratic muscular twitches. He could protrude the tongue, and follow the finger with his eyes, but when asked to do something quite different, he still protruded the tongue, or did something equally far from the mark. The aphasic condition previously referred to was now undeterminable owing to patient's cloudy general mental condition.

Temperature.—His temperature, prior to admission, had not been carefully observed, but from 11 P.M. on 20th May, when it was 101·8° F., it had steadily risen to 103·2°, at 6 P.M. on the 21st (the date of admission).

Pulse.—The records have never been higher than 84, indeed many of them were subnormal 56-58 and 60. He had a slight cough, but no expectoration was obtained for examination.

Bowels tended to be costive, bladder empty during the few hours he remained in the ward prior to operation.

The case was regarded as a complicated intracranial one arising from infective purulent otorrhœa. The restlessness, the nervous agitation, the occasional squint in the left eye, and frequent erratic muscular twitches, indicated a degree of lepto-meningitis. The high temperature, the repeated rigors, the pain in the upper third of the internal jugular and posterior cervical triangle, pointed in the direction of an inflammatory involvement of the sigmoid sinus with possible systemic invasion by pathogenic organisms. The slow pulse in presence of the high temperature, the mental obscuration and sluggish cerebration, coupled with the paresis of the members on the opposite side of the body, were in favour of encephalitis, probably purulent, and combined with the meningitis. The right-sided facial paresis of central origin and the paresis of the right arm, along with the slight aphasic indications, showed that the third frontal and the bases of the ascending opercular convolutions were implicated, probably from a lesion in the temporo-sphenoidal lobe, which localization was further supported by the state of the left pupil, which, though not widely dilated as when the third nerve is paralysed from pressure, was yet wider than its fellow and was fixed. An abscess in the temporo-sphenoidal lobe would have accounted for some of the symptoms. If this were present, the sudden increase of temperature noted as occurring two days before admission could have been caused by leakage of the infective pus into the ventricles, or on to the superficial brain membranes.

Operation.—The mastoid antrum was opened and found filled with pus; masses of granulation tissue bathed in fœtid discharge projected into the antrum from the middle ear. The middle ear was considerably enlarged, and there were minute necrotic portions of bone in its roof, some of which were removed, giving vent to extradural pus. The sigmoid groove was exposed, and a quantity of pus (amounting to several drachms) which lay between the dura and the skull was allowed to escape. The sinus was coated with purulent exudation, but blood still flowed through it. A circular disc $\frac{3}{4}$ inch in diameter was removed from the squamous portion of the left temporal bone, its lower border being $\frac{1}{4}$ inch above the posterior root of the zygoma, and in the same coronal plane as the external auditory meatus. When this portion of bone was removed, a small quantity of pus escaped. The dura mater was of a greyish yellow colour, and when cut into was quite soft, avascular, and appeared like a bit of sodden wash leather. It was fully $\frac{1}{8}$ of an inch in thickness. The underlying brain was of a slightly yellowish colour, and after removing a portion, about an $\frac{1}{8}$ of an inch in thickness, a mass of greenish brown, glistening œdematous foul-smelling slough was exposed, from which a dark coloured, semi-purulent fœtid fluid oozed. This mass was penetrated in various directions with a hollow needle, and although small quantities of a similar fluid escaped from the needle on each penetration, it was evident that instead of there being an abscess cavity, a large portion of the temporo-sphenoidal lobe was necrosed and in process of separation.

FIG. 49.—NECROSIS OF BRAIN.

Sagittal section of hardened brain through centre of left temporo-sphenoidal lobe, showing gap remaining after removal of slough (Case XX.).

To face page 118.

The aperture in the skull was enlarged anteriorly, and through the large aperture thus made, the slough which comprised the greater portion of the temporo-sphenoidal lobe was partially removed, care being taken not to encroach upon the living brain tissue.

It was evident that the descending cornu of the lateral ventricle had been invaded by the necrotic process, and the lateral ventricle was therefore freely opened. Subsequently, the cavity was carefully irrigated with a 1-40 watery solution of carbolic acid. During the operation there was no bleeding or even hæmorrhagic oozing seen from the brain tissue or membranes.

After operation patient was frequently dressed, and on each occasion considerable masses of slough and putrid discharge were still evacuated from the brain. His general condition was not much improved, with the exception that for a few days there was an increase in his intelligence sufficient to enable him to take some interest in the patients that were around him, and during which he also expressed himself intelligibly. On one or two occasions he used wrong words in a way resembling amnesic aphasia. With these exceptions he remained in a semi-conscious condition, lying either quietly, or in a state of muttering delirium. The left pupil was generally larger than its fellow, and the left eyeball had a slight external strabismus. The day after operation he had a cough and blood-streaked expectoration; it was noticed, however, that he frequently swallowed his expectoration, and as he grew weaker the expectoration seemed to be constantly swallowed. This was followed by looseness of bowels, and latterly by distinct diarrhœa, the stools being of a dark greenish colour with a foul odour resembling that emanating from the brain. On the 23rd and 24th he had two distinct rigors with subsequent perspiration.

On auscultation large coarse râles were found with inspiration and expiration, more especially over the right base behind.

His temperature continued high, reaching over 104° F., and it was evident that he was becoming exhausted by the infective process in his brain, lungs, and intestines. He died on 29th May, 1891, the 29th day of his illness, a little over six days after operation.

Post-Mortem Sixteen Hours after Death.—The longitudinal sinus had about its middle a firm pale clot connected with a couple of thrombosed veins, which ran in the dura mater upwards from the temporo-sphenoidal lobe on the left side. Behind that, in the longitudinal sinus, there was an ordinary post-mortem clot. The dura mater over the whole extent of the temporo-sphenoidal lobe was in a softened condition. Part of it over the squamous and petrous bone was in a state of slough, the remainder being covered on its internal surface by a layer of semi-purulent plastic exudation, a very thin layer of which existed between the bone and the dura. The soft membranes over the brain generally were considerably congested; the congestion, however, did not amount to more than what is seen in the initial stage of lepto-meningitis. The veins of the temporo-sphenoidal lobe which were still visible, were filled with disintegrating blood clot. On the brain being removed, almost the whole temporo-sphenoidal lobe on the left side was seen to have been converted into disintegrated

slough, only a thin superficial rim of recognizable brain tissue covering the basal and upper portions, while the gap in the external aspect towards the temporal bone revealed the disintegrated mass within. This mass communicated freely with the left lateral ventricle. Practically the whole of the temporo-sphenoidal lobe, except this thin covering, had disappeared.

The sigmoid sinus was covered with granulation tissue at the part exposed during operation, and there was considerable dural congestion surrounding the sinus. The bone of the sigmoid groove, especially at the knee, was deeply stained of a dark bluish brown colour. The cerebellum, at the part over which the sinus lay, was likewise stained of a blue colour, and its surface at that point was somewhat softened. These appearances, as far as the external aspects of the brain are concerned, are seen in the photographs.

The examination of the temporal bone with its attached soft parts showed, that there was an erosion in the tegmen tympani, as was discovered at the operation, and on the tegmen tympani inside of the petro-squamosal suture there was a necrosed portion of bone about a quarter of an inch in breadth, nearly half an inch in length. This necrosed portion was in process of separation, and in the periphery of it there was granulation tissue communicating with the interior of the middle ear and antrum. The dura mater over this part was covered with a thick coating of pus, formed into an adherent layer. The internal jugular had its walls thickened, and was of a buff colour; the soft tissues in its vicinity were deeply injected of the same dark blue colour as was seen in sigmoid groove. The external coats of the internal carotid artery and the coverings of the nerves were also deeply injected, as were likewise the soft tissues and bones surrounding the cervical venous plexus, and the posterior and anterior condylar veins.

State of Lungs.—The lungs were pneumonic, with one or two wedge-shaped infarctions at their less inflamed parts. There were old pleuritic adhesions.

Remarks.—It was evident that all this mischief had arisen from the chronic otitis media. The small portions of necrosed bone in the middle ear were undergoing separation, but meanwhile the pus associated with the disintegrating process necessary for their removal had become infective and had set up cerebral necrosis, probably through infective venous thrombi. It is probable that although this slough had formed, the temperature still remained low as long as it was a localized process, but when some of the infective products had found their way into the lateral ventricle, the temperature had rapidly risen. This takes place frequently when an abscess bursts into the ventricles, but when this event occurs the rise in temperature is generally accompanied by evidence of respiratory difficulties, rapid increase of pulse, and often by profound coma, these phenomena being occasioned by the larger quantity of the invading material suddenly set free from the abscess cavity and poured into the ventricles. In this case the mobility of the infective matter was slight, its ingress to the ventricle being limited by the slough, which acted as a barrier. Besides, the local

infective products had reached the blood stream and gained an entrance into the lungs, probably by way of the sigmoid sinus, causing infective pneumonia and infarction, and from the expectoration which was swallowed by patient it is probable that the diarrhœa was occasioned; although this might also have been due to infective organisms or their products brought through the blood acting on the intestines as is frequently seen in toxæmia.

The paresis of the opposite side of the body is explained by the meningeal extension of the inflammation from the temporo-sphenoidal lobe into the motor area, and if so, the speech centre might likewise have been involved by extension of the lepto-meningitis. There was no visible naked eye lesion in these parts of the brain. It is possible that the motor strands might in this case have been implicated through the inflammation extending to the internal capsule, or from within the lateral ventricle. The chronological order in which the motor paresis had occurred prior to admission had not been noted, therefore it was impossible to determine whether the paralysis was due to a cortical lesion or one in the motor strands.

The cerebral necrosis could be accounted for by an embolism of one of the middle cerebral arteries, but the appearance of the necrotic tissue was darker than is generally seen in anæmic cerebral necrosis, though this change in colour might have resulted from subsequent disintegrating process in the presence of infective matter. The main branches of the left middle cerebral artery were examined but no embolus was found. Some of the smaller arteries and veins were unfortunately obscured or lost in the disintegrating process, therefore their state could not be ascertained. On the other hand there was abundant evidence of extensive venous thrombosis over the temporo-sphenoidal lobe, and two large superficial trunks passing over the surface of the brain from the Sylvian fissure to the longitudinal sinus were thrombosed, and had evidently been the means of carrying the infective thrombosis into the longitudinal sinus. The colour of the necrotic brain area corresponded better with venous thrombic ætiology. Figs. 47, 48, and 49 illustrate the gross appearances.

Pyæmic abscess of the brain may arise from infective matter originating in some source of infection in any part of the body. This peccant matter entering the blood is carried by the blood-stream until it is deposited in a small vessel producing infective embolism. The diseases which have been known to give rise to this condition in the brain are, among others, disintegrating pneumonia, fœtid bronchitis and empyema, fœtid pericarditis, infective compound fractures, seldom acute infective periostitis, and occasionally infective ulcers of the intestines and abdominal cavity. Infective peritonitis generally ends fatally so soon after its onset that cerebral abscess has no time to form. Böttcher[1] relates a case where a cerebral abscess developed

[1] Virchow, *Jahresbericht*, 1869, p. 51.

subsequently to lung suppuration, and lung pigment was found in the abscess of the brain. There are two cases reported of oidium albicans[1] which formed in the mouth and pharynx, producing cerebral abscess, in one of which the brain was studded with small abscesses filled with oidium albicans.

It is well to remember that pyæmia, though generally the cause of multiple abscesses, may itself arise from intracranial suppuration, especially when the sinuses are also involved.

Multiple abscess.—Abscesses are rarely multiple saving when they originate in pyæmia, in which case they are in two-thirds of the cases multiple, and generally very numerous, and invade both brain and cerebellum. In one case of pyæmia seen post mortem by the author, there were four large abscesses, two on the left side in the occipital lobe, one in the occipital lobe on the right side, and one in the left parietal. There were besides many small ones about the size of beans, and the brain tissue was softened and œdematous round each of these. 93 per cent. of abscesses from traumatism are single: 87 per cent are single when due to otitis media.

On several occasions the author has found an abscess in the temporal lobe and pus in the cerebellar fossa, both inside and outside the dura mater, each arising from the same source, otitis media. Dr. M'Conville of Glasgow mentions the fact that he found on post-mortem examination of one of his patients an abscess in the temporo-sphenoidal lobe, and also one in the cerebellum. In one of author's cases (Case XXXVII.) an abscess was found involving the posterior aspects of the temporal lobe, and the anterior aspect of the occipital lobe as well as one in the cerebellar fossa.

Parts of the brain most apt to be affected by abscess.—While abscesses may form in almost any part of the brain, they are chiefly located in the white matter. Ulceration does occur in the superficial layers of the brain and its membranes, so that superficial abscess forms between the brain and dura mater. When the abscess originates in injury, the frontal and parietal lobes are the most frequent seats: when from otitis media, the temporo-sphenoidal lobe and the cerebellum are the parts usually affected. The pons Varolii, the corpus striatum, and the optic thalamus, have been the seats of abscess, while Abercrombie mentions an instance occurring in the medulla oblongata. Pyæmic abscesses are found

[1] Wagner, *Jahrb. f. Kinderheilk.*, i. p. 56.

in many different parts of both cerebral hemispheres, but the occipital lobes are more frequently affected than others.

Statistics show that abscess occurs in the cerebrum about four times as often as in the cerebellum. The author, however, questions the accuracy of the data, as his experience indicates a greater proportion of cerebellar abscesses. Abscess is rare in the pons and medulla oblongata. It affects both sides of the cerebellum with equal frequency, and is very rare in the middle lobe.[1]

Mode of healing of a cerebral abscess after evacuation.—After an abscess has been evacuated the brain tissue tends to fill the gap left by the removal of the pus. If it be an acute abscess, the resiliency of the brain is retained and its expansion is rapid, a few minutes to a few hours sufficing to close the gap. After evacuation of chronic encysted abscess, the expansion is much slower, taking days before the cavity is obliterated. When the wound has been at once closed after evacuation and the dura replaced, there is a probability of the brain remaining free from adhesions, but as a rule it becomes more or less fixed to the dura. When the wound has to heal by granulation tissue, the opening into the brain meanwhile being kept patent for pus evacuation, the pia mater adheres to the cicatrix, soldering the brain to what becomes a rigid wall. Such anchoring of the brain is subsequently apt to produce a shock on sudden movement of the patient, as on sudden rising from a recumbent or sitting posture. This physical effect directly applied to the brain itself, though acting like a drag or pull, is akin in its physiological result to a blow applied to the cerebrum. It is apt to cause unconsciousness, generally of very brief duration. The man may fall, but is able to pick himself up in a few seconds. In some few instances the unconsciousness lasts longer. When very frequently repeated, even when the effect is so slight as to be only slightly perceptible to the patient, this frequent dragging is apt, months or years afterwards, to produce encephalitis over an extended area of the brain. This cerebral irritation might lead to epileptic fits, though the author is not aware of any undoubted instances following operation for cerebral abscess. There are,

[1] Gowers' *op. cit.* gives from compilation—

186 in cerebrum (not all separate cases).
41 „ cerebellum.
3 „ pons.
1 „ medulla oblongata.

however, many such cases resulting from traumatisms, in which the brain has been soldered to the unyielding parietes.

Occasionally a depression, which is filled with serum, rarely with cerebro-spinal fluid, remains in the brain surface over the site of a former abscess. The site of a small brain abscess previously evacuated is difficult to recognize. There may be a mere puckering of the brain fibres as was seen in Case No. XI.

TUBERCULOSIS AND CARCINOSIS OF THE MIDDLE EAR.

There are two non-pyogenic diseases affecting the middle ear and its annexa—tuberculosis and carcinosis—to which a reference may be conveniently made at this place.

TUBERCLE OF MIDDLE EAR.[1]

Tubercular ulceration, originating in the middle ear and affecting the mastoid and petrous bones, is frequently met with in infancy and childhood. It is a very insidious affection, often occurring without rupture of the membrana tympani, with no pain or only a little of an indefinite character.

The friends first become anxious from the evident deterioration of health without apparent cause. The seat of disease is often overlooked, as the function of the organ—hearing—is often fairly preserved. The sub-mastoid and sub-occipital glands are frequently enlarged, and at a later stage those which follow the course of the internal jugular become affected. When the disease has invaded the Fallopian canal, facial paralysis ensues which at once draws attention to the region. Destruction of the bone is frequently very great before there is evidence of inflammation over the mastoid externally. In some cases, only a thin shell of the petrous and mastoid is left, the whole interior being filled with masses of granulation tissue containing caseating patches. There may be very little purulent discharge, the bones being eroded by caries sicca. The disease is not infrequently found on both sides of the head, both mastoids and both petrous bones being affected. Occasionally the tubercular invasion involves the whole of the base of the skull, the tubercular infiltration of the petrous and mastoid

[1] Nathan (Deutsch. Archiv. für klin. Medicin, Bd. 35), found tubercle bacilli in the pus from 12 out of 40 cases of otorrhœa. In 8 of these tuberculosis of other organs was present.

on the one side, communicating through the basi-sphenoid and basi-occipital with a similar infiltration in the petrous and mastoid on the other.

From this diseased focus tubercle occasionally spreads through the tegmen tympani or petro-squamosal sutures to the brain membranes, producing lepto-meningitis. When this occurs early, before much destruction of the bone surrounding the middle ear has occurred, and before the membrana tympani has been ruptured, the primary focus is apt to be overlooked, and the cases are then described as ordinary tubercular lepto-meningitis.

Abscess of the brain seldom follows tubercular disease of the structures surrounding the middle ear, partly owing to the susceptibility of the inner membranes to the dissemination of tubercle, and the rapidly fatal character of tubercular meningitis, and partly owing to the free outlet for discharge frequently formed by the extensive tubercular destruction of the osseous walls. When abscess does occur in such cases it is generally superficial and in immediate proximity to the tubercular perforation. Frequently, this superficial cerebral abscess communicates with the middle ear by a minute passage through the granulation tissue surrounding the osseous perforation. Instead of a distinct abscess in the midst of the cerebral tissues, a superficial cerebral ulceration is occasionally found in connection with tubercle of the middle ear, the pus from this ulceration being confined by the soldering of the meninges at the periphery of the ulceration to the base of the brain and the dura respectively. Such a purulent collection might thus appear as a localized cerebral abscess, the pus being retained between the brain which formed the dome, and the dura which formed the floor of the abscess cavity.

The sigmoid sinus is not infrequently exposed by tubercular erosion of the sigmoid groove. This may occur long before there is any evidence of thrombosis of the sinus.[1] Thrombosis may occur in such a case from gradual invasion of the sinus by tubercular granulation tissue, or an acute infective process may

[1] Several instances of this kind have come under the author's observation. The excavated mastoid has been exposed by removing its outer shell, the whole cavity found filled with granulation tissue, a portion of the sigmoid covered with granulation tissue being exposed by osseous erosion. On one occasion almost half of the circumference of the sinus was thus exposed, and it was so thickly covered with granulation tissue springing from its surface, as to be somewhat difficult to recognize. The possibility of damaging the sinus while removing the masses of granulation tissue is obvious, unless such a pathological condition is borne in mind.

be superimposed upon the tubercular one, occasioning infective thrombosis. It must be borne in mind that degenerating and liquefying tubercular foci present highly favourable nutrition for the cultivation of pyogenic organisms, and, therefore, the infective processes set up secondarily from such sources are very virulent.

In connection with tubercle of the middle ear, antrum, and mastoid, enlarged cervical glands are common. Though the sub-mastoid and sub-occipital superficial glands are generally enlarged, those surrounding the internal jugular are often affected, and it is much more serious if they take on an inflammatory action. When such glands become enlarged a careful examination of the middle ear and mastoid regions ought to be made. If the membrana tympani be intact, inquiry into pharyngeal discharge may reveal a constant secretion from that region. Posterior rhinoscopy may show pus emanating from the orifice of the Eustachian tube. Even when no such evidence is obtained this does not preclude the possibility of tubercular invasion of the mastoid and petrous regions, as that may occur without pus production. A careful test of the function of the affected ear will generally help in the diagnosis. It must be borne in mind that patients with extensive granulation masses in the antrum, mastoid cells, and even in the middle ear, can hear fairly well for ordinary purposes, and yet marked difference in the acuity of acoustic perception may be detected. On the other hand, the mastoid region, including the antrum, may be invaded with tubercular granulations, without the middle ear participating to any great extent in the process.

It is of little use to remove enlarged cervical glands without first removing the cause of their enlargement or ascertaining that it has already been eradicated.

CASE XXI. *Abstract of a case of tubercle of the tympanum and petrous bone, with invasion of the brain membranes through the petro-squamosal suture.*

The writer was consulted by a medical friend regarding one of his children, an infant of six months, who had been falling off in flesh and was becoming very weak without perceptible cause, saving a series of enlarged cervical glands situated in the right sub-mastoid region, and also running parallel to the internal jugular, probably on the carotid sheath. There was no detectible pain on touching the glands. The patient exhibited a general fretfulness and malaise. There was supposed to be no aural discharge, but

minute examination showed that the right membrana tympani, which lay very obliquely horizontal, was thickened, and a little bud of granulation tissue protruded at its edge. There was a slight muco-purulent discharge in the inner part of the external ear. Over the corresponding side of the face there was a slight shade of passivity, indicative of an involvement of the facial, probably through the tympanic aperture in the aqueductus Fallopii, generally present at this early age. The lesion was regarded as probably tubercular, situated in the middle ear, and invading the growing bone from that point. The glands were secondary enlargements. The little patient was too feeble to permit of surgical interference, and soon after developed lepto-meningitis from which she died.

The post-mortem showed serous lepto-meningitis at an early stage without any detectable tubercles in the pia mater, which was, however, firmly adherent to the dura over the tegmen. At this point the dura was of a bright red colour, and scattered over its surface were a series of tubercles arranged in two crescentic lines, the one above and external to the petro-squamosal suture, the other on its inner side over the tegmen. These proved to be tubercular invasions of the dura through the petro-squamosal suture directly continuous with the tubercle in the middle ear. Before placing the specimen in spirit, these tubercles presented a beautiful appearance like crescentic rows of glistening pearls on a rose-coloured base. In the cancellated tissue of the mastoid and in the cavity of the tympanum, there was granulation tissue and caseating matter, at the periphery of which tubercular giant cells were found. The brain was healthy. The cervical glands in the sub-mastoid and sub-occipital regions, besides those running in line with the internal jugular, were enlarged. On section they were found to be distinctly tubercular.

The lungs and peritoneum were normal. The intestinal glands were slightly enlarged.

Remarks.—This case was one in which the tubercle (possibly due to the cow's milk on which the child was fed being probably tuberculous) formed at a very early period in the tissues surrounding the middle ear, and in the spongy portion of the mastoid. From these the disease invaded the dura mater, through which it penetrated and set up the lepto-meningitis.

Précis of an instance of tubercular ulceration of base of skull.

At a post-mortem examination of a man (C. P. O., 1880), aged 45 years, who was markedly tubercular, the lungs being filled with large caseating masses, the bones of the base of the skull were of a bluish appearance, from the contained masses of granulation tissue, showing through the white bone. So thin were these bones that pressure with the tip of the finger caused the internal table to crumble and to expose the granulation tissue between the two tables of the bone. This was especially apparent over the basi-sphenoid and basi-occipital, and both petrous and mastoid bones. Both membranæ tympanorum were intact. There was a slight amount of pus in both middle ears, and a little in their corresponding Eustachian tubes. Patient

died from tubercular exhaustion, but had been doing business until a month before his death. The dura at various points at the base was eroded, and was generally soft and pulpy. There was adhesion of the soft membranes to it, and there was chronic tubercular lepto-meningitis.

CARCINOMA OF THE MIDDLE EAR AND MASTOID PROCESS.

Carcinoma of the middle ear, mastoid and petrous bones is uncommon. Like all carcinomata, it generally affects people in advanced life. It gives rise to severe lancinating pains, aggravated on exposure to cold. These pains are at first confined to the ear, then the branches of the fifth are involved, subsequently the pains shoot over the whole of the same side of the head, and finally they penetrate to the opposite ear. These may exist for months before there is any external appearance of the carcinoma. Facial paralysis is prone to arise from invasion of the aqueductus Fallopii. The appearance of the patient is, however, indicative of the presence of some severe malady rapidly reducing the strength, and in virulence out of proportion to the apparently limited area affected. He soon becomes emaciated and exhausted. The cervical glands in proximity to the internal jugular are enlarged, and either through them, or possibly through direct contamination of the sigmoid sinus, the liver and possibly the lungs become involved by secondary deposition. The carcinoma invades the antrum, the epithelial masses forcing themselves into the mastoid cells and eroding their walls. The carcinoma may appear externally behind the ear over the mastoid process, or it may penetrate the walls of the external auditory canal, or protrude through the membrana tympani. It appears then in the form of dense hard masses—epithelioma, accompanied by a foul-smelling purulent discharge. The temperature remains about normal, unless the discharges become infective, when secondary pathogenic infection follows. Infective thrombosis of the sigmoid sinus is apt to occur in such cases, and from a similar source lepto-meningitis may ensue. The mind of the patient is quite undisturbed except during the painful paroxysms.

It is unlikely that such cases could be mistaken for cerebral abscess or meningitis. The violent otalgia and cephalalgia, if taken alone, might resemble the outbreak of an acute cerebral affection, but the duration of the disease would negative the latter. If any doubt existed concerning the diagnosis, the removal of a small portion of the tumour—granulation tissue?—for microscopical examination would settle the matter.

Treatment.—When the disease is early detected, it might be removed by ablation of the mastoid and middle ear. When it has become disseminated through the mastoid, and especially when it has involved the petrous portion, and infected the glands surrounding the internal jugular, it had better be left alone and treated with sedatives.

Précis of three cases of carcinoma of middle ear.

The author has seen three cases of carcinoma of the middle ear and mastoid.

Case XXII. The first, a lady, aged 60, seen in consultation with Dr. Barr. The external auditory meatus was already involved. The mastoid process was tender to the touch, and the growth which sprouted from the external auditory canal was of almost cartilaginous consistency, and bathed in septic pus. She suffered great pain, and was rapidly emaciating.

Case XXIII. The second, a woman about 56 years, was admitted into the Royal Infirmary. The growth already protruded through the posterior aspect of the external auditory meatus. She suffered excruciating agony along all the branches of the seventh and many of those of the fifth. The mastoid process was opened by operation, when it was found to be filled with dense masses of the neoplasm which had already involved the walls of the sigmoid sinus, whose lumen was occluded. The antrum and middle ear were exposed and their carcinomatous contents cleared out. The mastoid process was ablated. This gave great relief for a couple of months. The parts then became refilled with the neoplasm, and the old pain returned as before.

Case XXIV. The third patient was a man, aged 66 years, who was admitted into the Royal Infirmary. His tumour had already extruded behind the ear through the mastoid, and also through the membrana tympani. The involved parts were dense and hard to touch ; they were found on microscopic examination to be epitheliomatous. His pain was not so severe as in the last case, and he described it as a constant gnawing, especially referred to the branches of the fifth, from which it was inferred that the carcinoma had penetrated inwards, through the petrous bone, as well as backward over the mastoid. There was an oozing of putrid pus from the ear. He had symptoms of infective thrombosis of the sigmoid sinus with lung complication. The internal jugular was eroded, and its upper two thirds was surrounded by enlarged glands. The liver was hard and nodular, probably from carcinomatous infiltration. No operation was performed.

Had such cases been presented at an early period their eradication would have been more hopeful. Carcinoma of the epitheliomatous or scirrhus variety implicating the skull, though accompanied by fœtid and sometimes infective discharge, seldom leads to infective intracranial inflammation, probably on account of the dense neoplastic barrier interposed between the healthy tissue and the infective organisms.

CHAPTER III.

SYMPTOMS OF ABSCESS OF BRAIN.

The course of abscess of the brain, with its accompanying phenomena, may be conveniently divided into three stages, each differing markedly from the others. The first includes the initiatory symptoms, the second those of the fully formed abscess, while the third embraces those connected with the various terminations of that affection when uninterrupted by treatment.

The initiatory stage.—In the great majority of cases other than traumatic or pyæmic, the patient has had a chronic purulent discharge from the middle ear, often dating from an attack of one of the exanthemata in childhood, or he has had a chronic ulceration about the nose or mouth. The following is the course of brain abscess arising from chronic otitis media.

In some instances, especially among the poorer classes, patients deny that they have had a discharge from the ear, but on minute inquiry admit that a little moisture may have issued occasionally from the external ear, though they had paid little attention to it. In others, though unaware of ear disease, or of purulent discharge from the ear, aural examination reveals the presence of otitis media of a chronic kind in one or other ear. Otitis media may exist and produce serious and even fatal results, without perforation or destruction of the membrana tympani.

The otorrhœa may have given little trouble, and its long continuance without apparent harmful result may have lulled the initial fears, till it was regarded as of no importance.

A person affected with chronic otitis media becomes ill somewhat suddenly, often after exposure to cold, sometimes after a blow or knock on the head, and occasionally without any direct assignable cause.

Pain in the ear is first experienced, variously described as hot, burning, or shooting in character. It is at the outset confined to the ear, but soon extends into the temporal area, and after the first twenty-four hours, though the pain is principally localized in that region, yet it radiates to the frontal and occipital. Even when the abscess is located in the cerebellum, the pain may be referred to the forehead. Occasionally there are shooting pains, which the patient describes as darting through the brain from one ear to the other, though these are more often met with in sigmoid sinus thrombosis. It is said that one patient complained of pain on the left side of the head, while the abscess was on the right side. The author has not met with any such case.

The pain is usually so severe that the patient cries out and rolls in agony, and describes it as excruciating. It may be either continuous or intermittent. Occasionally there are violent neuralgic paroxysms, with dull aching sensations in the intervals.

Vomiting during this stage is frequent, and bears no relation to the taking of food—patient usually being unable to do so—or to any gastric derangement. The vomiting may be quite unaccompanied by nausea.

Rigors.—A rigor is one of the most constant early symptoms. It may be of the most varied degree, from the mere feeling of cold, which cannot properly be designated as a rigor, to the most violent shivering. It is sometimes so slight as to escape detection by untrained observers. The patient may have experienced a slight sensation of cold, a shrugging of the shoulders and a tendency to chattering of the teeth. The *cutis anserina*, however, is well marked at this stage. When the patient's friends do not describe shiverings as having occurred, they may be able to say that he has complained of feelings of cold. In other instances, the rigor is so pronounced, the shaking so violent, and the surface of the patient's body assumes such a livid pallor, that the friends not unfrequently regard the attack as convulsive. In one such instance the patient's friends designated it as a convulsion, but the medical attendant, who happened to see the rigor, rightly described it. The rigors vary not only in intensity, but likewise in duration, lasting from a few minutes to half-an-hour, or longer. Though repeated rigors may occur in cerebral abscess, yet this is not to be regarded as a feature of uncomplicated cases. The more often the rigor is repeated, the more likely is the abscess accompanied by some form of systemic infection. Repeated rigor

is a marked feature of infective sigmoid sinus thrombosis, especially when followed by infective pulmonary infarction. When the latter occurs the rigors are very numerous, and are more frequent towards the fatal termination. Rigors have been stated to have occurred with such regularity—one every twenty-four or forty-eight hours—as to simulate ague. It is probable that such cases have either occurred in persons previously the subject of ague, or they have been suffering from some complication, and not from cerebral abscess alone. In one instance, under the author's care, the vomiting, the rigor, and the convulsion followed each other with such regularity that there were exactly twenty-four hours between each.

The *temperature*, during this preliminary period, in the few cases where it has been accurately observed, has been slightly above normal. The circulation is accelerated. The tongue is coated and furred. The patient's general condition is that of prostration with occasional pains in the limbs.

As a rule, the *secretion of pus from the middle ear ceases* or becomes much less in quantity during this initial period. It may have been flowing profusely before the attack and have somewhat suddenly ceased. The friends of the patient frequently say they have known him to have had somewhat similar attacks, which terminated as soon as the otorrhœa reappeared.

It must not be forgotten that symptoms such as pain, vomiting, vertigo, and rigors, with fever, often usher in suppurative otitis media, without any intracranial involvement, and again cases of cerebral abscess do occur where, without the initial pain, the patient passes into a lethargic condition, accompanied by general malaise, with low temperature, slow pulse and respiration.

This initial stage is of variable duration, from twelve to seventy-two hours, or even a week. At its termination the patient passes into a state in which all his senses seem dulled.

SECOND STAGE.—This is the period in which most cases of cerebral abscess are brought under the surgeon's notice. He seldom sees any in the first stage, though he unfortunately sees many in the third. In the second stage all the patient's senses have become more or less blunted.

The *former pain* and suffering experienced by the patient have become greatly assuaged, partly because of the diminution of the pressure which produced the pain, and partly owing to the patent fact that encephalitis has deadened his sensibility.

The patient may still complain of pain over the ear and head, but its acute edge has been removed, and it is no longer the dominant symptom. It may not be spontaneously complained of, and only on minute inquiry may its existence be determined. The patient often lies with his hand over the affected part, and in some cases moans from pain, but as a rule the patient is in a soothed condition, often attempting a smile when spoken to, and tends to lie quiet and doze.

Percussion over the affected mastoid process and the squamous part of the temporal, and on same side of the head, frequently makes the patient wince and apparently aggravates the pain, though it does not affect him when applied to the opposite side of head. The one side ought always to be compared with the other in estimating the significance of percussion.

In abscess in the temporo-sphenoidal lobe or cerebellum, when accompanied by sigmoid sinus thrombosis, the sterno-mastoid muscle is often rigid, the subjacent tissues painful, and pain is complained of along the course of the internal jugular. In such cases further pain may be elicited on deep pressure in the apex of the posterior cervical triangle. (On this point see further under "Chapter on Sinus Thrombosis.")

Owing to *slow cerebration* the mode of answering questions is peculiar. When a question is asked, the patient does not at once answer, as an ordinary person would. He pauses, and looks vacantly forward through dreamy eyes. Just as the interrogator is about to repeat the question, believing he has not heard, or having heard has not understood, the patient answers slowly, briefly, and, as a rule, correctly. At a later stage, the answers may become monosyllabic, however incomplete or inadequate such answers may be; as if patient, though willing, found himself unable to put forth the necessary energy to do more.

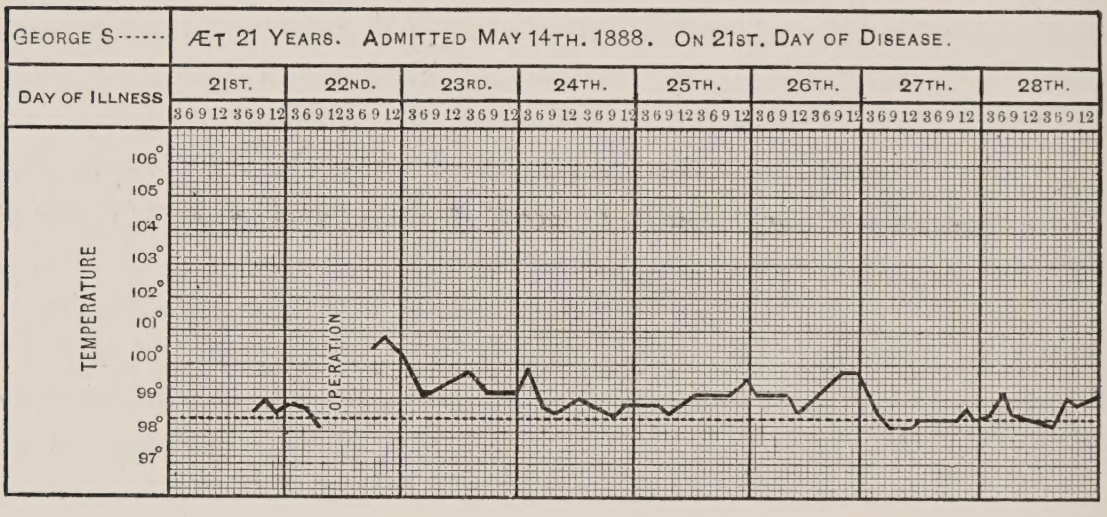
Accompanying the slow cerebration there is a marked *want* of *sustained attention.* The patient may be roused from his somnolent condition into a sufficiently wakeful state to understand and answer simple questions. During a long interrogation, however, he will drop asleep, or even when he attempts—which he seldom does—a long answer, the first part of it will be delivered correctly, but as he proceeds, the utterance becomes more and more slow and less articulate, the look in the eyes more distant, the eyelids close, and he sinks again into the dozing condition from which he had just been aroused. The patient may ask for a drink, and before the

vessel is lifted from the table and placed to his lips he falls asleep, and has to be rewakened before he will drink. This pathological sleep is never sound, the patient never looks refreshed. Still later, the cloud of *mental obscuration* deepens until it is difficult to rouse the patient sufficiently to enable him to answer the simplest questions, or to take the necessary fluid food.

Along with this failure of mental energy there is an accompanying *gradual diminution of the ability to apply his strength.* The power still exists, but the will to exert it is slight or altogether in abeyance.

The slow cerebration, want of sustained attention, mental obscuration, and tendency to doze, along with the failure in the application of strength, all closely resemble the same phenomena

CHART C.—ABSCESS OF BRAIN.



The temperature chart of a typical uncomplicated case of cerebral abscess.

exhibited in cases of opium poisoning; but in the latter, all the stages are gone through within a few hours. The likeness is also observed in the slow pulse and respiration, and in the depression of temperature.

The *temperature* is generally about normal, or slightly subnormal, 97° F. to 99° F. This is very typical of uncomplicated cerebral abscess, and is in marked contrast to phlebitis, sinus thrombosis, and to the majority of meningeal cases, especially suppurative lepto-meningitis, such as arise from infective otitis media. The fact of the temperature being about normal is apt to throw the uninitiated off their guard, and they express surprise that cerebral abscess can exist with a normal temperature (see Charts for comparison). After evacuation of the abscess, the

temperature rises quickly to over 100° F., and then gradually falls to normal.

The *pulse* is strikingly cerebral, slow and full, though sometimes weak and soft. Fifty to sixty per minute is usual, though it may be as low as thirty per minute.

The slow pulse is a common feature of certain cerebral affections, such as the encephalitis which accompanies tumour as well as abscess, but it is also found in excessive intracranial pressure, such as may be exerted in both these diseases as

CHART D.—INFECTIVE THROMBOSIS OF CAVERNOUS SINUS.

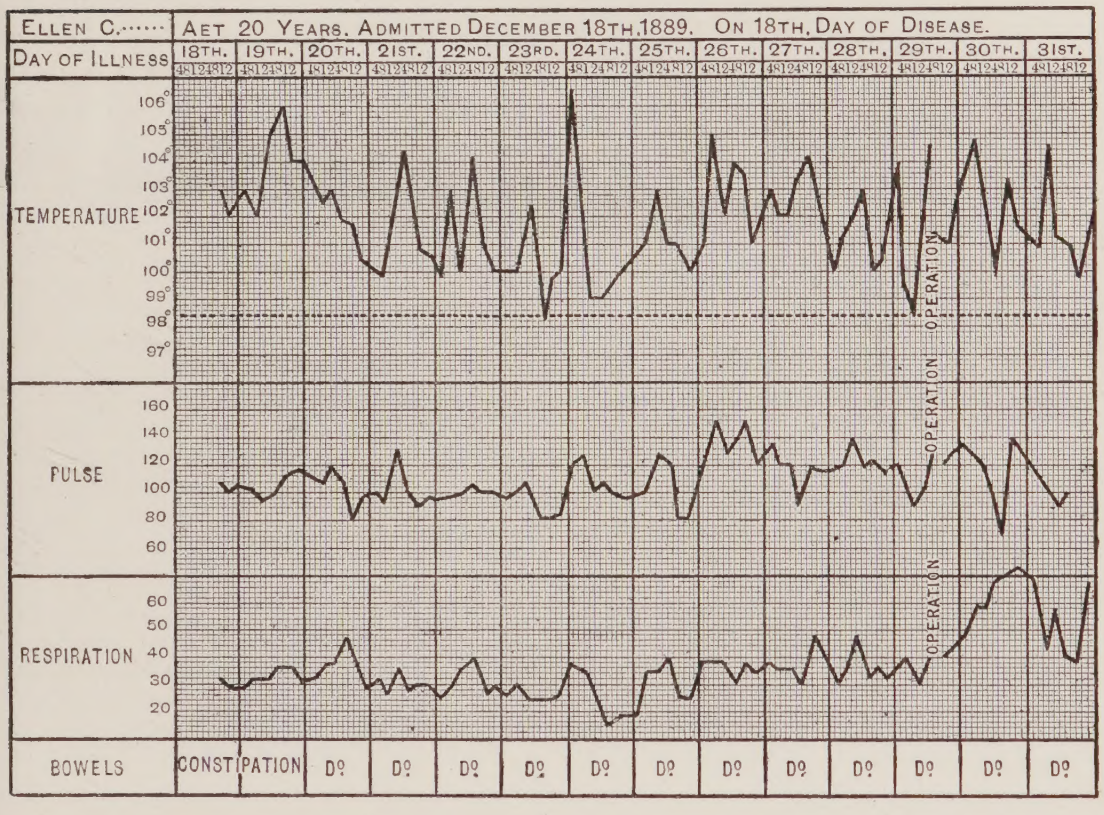

ELLEN C.·····
AET 20 YEARS. ADMITTED DECEMBER 18TH, 1889. ON 18TH. DAY OF DISEASE.
DAY OF ILLNESS
18TH. 19TH. 20TH. 21ST. 22ND. 23RD. 24TH. 25TH. 26TH. 27TH. 28TH. 29TH. 30TH. 31ST.
4 8 12 4 8 12 4 8 12 4 8 12 4 8 12 4 8 12 4 8 12 4 8 12 4 8 12 4 8 12 4 8 12 4 8 12 4 8 12
TEMPERATURE
106° 105° 104° 103° 102° 101° 100° 99° 98° 97°
OPERATION
PULSE
160 140 120 100 80 60
OPERATION
RESPIRATION
60 50 40 30 20
OPERATION
BOWELS
CONSTIPATION Dº Dº Dº Dº Dº Dº Dº Dº Dº Dº Dº Dº

Chart of infective thrombosis of cavernous sinus, for comparison with that of cerebral abscess.

well as in others. It is occasioned by large extradural clots of blood as they occur in cases of fractured skull. As a point of general diagnostic importance, when a slow pulse accompanies a high temperature, it points strongly to an intracranial affection, to the exclusion of a general systemic disease. When the same phenomena are met with in the presence of purulent otitis media, which has induced intracranial disease, they point to meningitis accompanied by encephalitis or cerebral abscess. In the acute meningitis originating from otitis media or from infective wounds, the pulse is quick and small. It is

likewise rapid in phlebitis of the sinuses and in infective thrombosis (see Charts).

The influence exerted on the pulse rate by encephalitis and pressure respectively is difficult to estimate. On the one hand small abscesses do produce distinct lowering of the pulse, and it is true that its rate is not decreased in *exact* ratio to the increase in size, as a patient with a cerebral abscess containing

CHART E.—INFECTIVE PURULENT CEREBRO-SPINAL LEPTO-MENINGITIS.

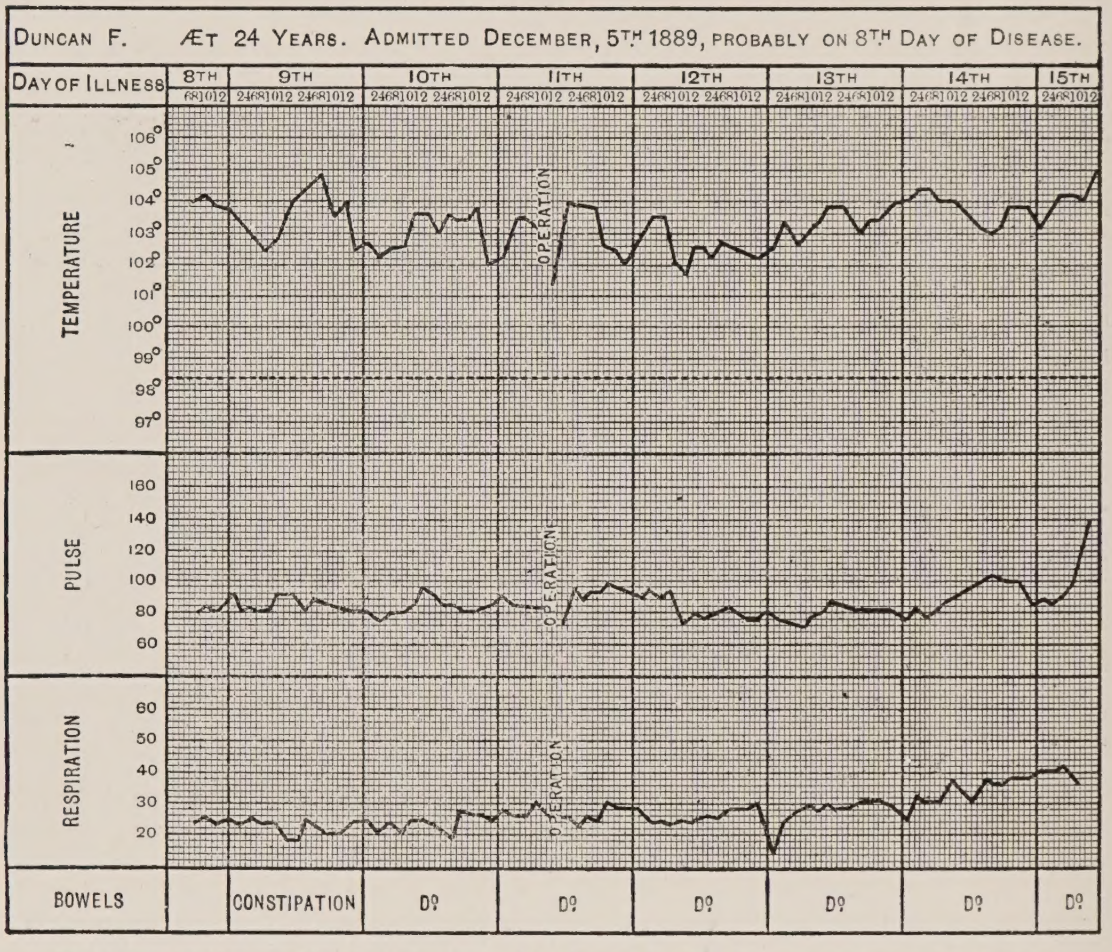


Chart of infective purulent cerebro-spinal lepto-meningitis, for comparison with that of cerebral abscess.

an ounce may have just the same pulse rate as another with an abscess containing half that quantity. On the other hand, a large uncomplicated abscess is generally accompanied by an appreciably slower pulse rate than a small one. In abscesses observed during their period of increase, the pulse rate shows a diminution as the abscess grows. Again, a very striking acceleration of the pulse is witnessed at the moment when a large abscess is evacuated, and before a change in the neighbouring encephalitis could possibly be effected. It is therefore probable that the pressure in such cases is the chief cause of

the slow pulse rate. It must be borne in mind that in some instances of cerebral abscess there may be little increase in the intracranial pressure, as the pus occupies little more than the area left by the molecular disintegration, and in such cases the pulse rate may be only slightly affected (see Charts of temperature, pulse, and respiration).

The *respirations* also are slow, though usually regular. They are sometimes as slow as eleven per minute. In cerebellar cases the respirations are generally much slower than in abscess of the cerebrum, and often they are irregular, and occasionally of a Cheyne-Stokes character. In pressure on the cerebellum affecting the pons, the respirations may cease long before the heart does. In one instance, observed by the author, of intradural effusion, into the cerebellar fossa, of blood which had also found its way into the fourth ventricle, the breathing ceased first. The heart continued to beat for five minutes afterwards, gradually growing less powerful, but regaining its vigour on the commencement of artificial respiration. The latter was maintained for ten minutes, in hope of re-establishing the respiration, without attaining that result. All this time, however, the heart continued to beat, and a slight pulse could be felt even at the wrist.

In a case reported to the author by the medical men in charge, a somewhat similar arrest of respiration occurred. It was one of cerebellar abscess, which, however, was only discovered post mortem. For the purpose of opening the mastoid antrum, the patient had an anæsthetic, and while the operation was proceeding the respiration ceased, while the heart continued to beat quite regularly. Artificial respiration was begun, and was continued for twenty-four hours without eliciting any response in the form of natural respiratory effort. The heart continued to beat during the whole time the artificial respiration was maintained, and even for a few minutes after. At the post-mortem examination a cerebellar abscess of considerable size was found, which no doubt exerted pressure on the respiratory centre.[1]

In meningitis, the breathing as a rule is quickened, but when the cerebellar fossa is involved in suppurative lepto-meningitis the respiration may be slow and of the Cheyne-Stokes variety, —often three to five respirations alternated with a long pause,

[1] A case of a somewhat similar character occurred in the Liverpool Royal Infirmary, and is reported in the *British Medical Journal*, June 3, 1893. In that case the heart continued to beat for six hours after the arrest of voluntary breathing, artificial respiration meanwhile having been maintained.

lasting about fifteen seconds, and being terminated by a deep inspiration.[1] In thrombosis of infective form, the respirations are quickened.

Constipation is the rule, and it is often very obstinate.—In meningitis and tumour of the brain constipation is also frequent, while diarrhœa is the rule in infective sinus thrombosis.

The *urine* occasionally is retained, and requires to be removed by catheter. Overflow incontinence otherwise occurs. This is common to all cases of lowered cerebral function. In cerebral abscess the urine sometimes contains albumen, though not in marked quantity, when the kidneys are sound. The albuminuria passes quickly away after evacuation of the abscess. Polyuria and glycosuria have seldom been noted in cerebral abscess, probably from the infrequency of the location of the abscess in the pons or medulla oblongata.

Anorexia is the rule, the patient requiring to be encouraged to take his fluid nutriment.

Vomiting, which is so frequent in intracranial disease, and which occurs in the initial stage of abscess formation, is not a usual symptom during this period, especially when the patient lies in bed. If he attempts to move about in the upright position vertigo is induced, and vomiting often ensues. The vomiting, like that of all cerebral affections, is not associated with local gastric symptoms, and is usually not attended by nausea.

When vomiting is accompanied by persistent headache, it is always suggestive of intracranial disease. It occurs in meningitis, tumour, and in cases of intracranial pressure as well as in abscess. In the others it usually persists, whereas in abscess it is generally absent during the second stage. It does not bear any constant relation to the size or extent of the lesion as it may be produced by very small organic cerebral changes. Vomiting is attributed to the irritation produced upon the gastric part of the vagus centre, the central relations of the vagus being of wide distribution.

Vomiting is especially common in cerebellar abscess, and when in the second stage of abscess formation vomiting persists and is a prominent symptom, it is well to direct special attention to the cerebellum.

Convulsions are not a distinctive feature in cerebral abscess, nor do they occur very frequently. This opinion differs from that of Lebert who asserts,—probably basing his opinion upon

[1] Typical Cheyne-Stokes has gradually increasing respirations after pause.

statistical data,—that convulsions occur in one half of all the cases of cerebral abscess. It is evident that convulsions, and other phenomena of a like nature, will be present or absent, according to the particular area of the brain which is affected.

Convulsions seldom occur during the second stage in cases of abscess of the temporo-sphenoidal lobe. When they do, they are usually the result of secondary irritation of the motor area, or its conducting paths. Convulsive movements of an erratic kind accompanied by rigidity and occasionally by tetanoid movement, are apt to supervene when the pus from the abscess has leaked into the subdural space or into the ventricles. Abscesses seated in other parts of the brain are occasionally accompanied by convulsions, as occurred in an abscess seated in the white matter of the third frontal convolution (see Case XXXIV.).

When convulsions do occur and are carefully observed they are of importance as affording aids to diagnostic localization, especially when they are of a limited character. In this relation, however, it must be borne in mind that the convulsions may be the result of secondary irritation, the abscess being at some distance from the spot indicated by the convulsions. It must not be forgotten that severe rigors have been described by the friends as convulsions.

Paralysis may occur from destruction of the parts involved in the molecular disintegration at the seat of the abscess, or from pressure produced by the abscess upon the surrounding brain tissue, or from the enervating effects of the encephalitis in the zone surrounding the abscess. Paralysis is of considerable importance in local diagnosis, as its origin is generally close to the lesion, and must be produced either by pressure or be within the inflammatory zone surrounding the abscess.

The *colour of the face* is not of much diagnostic value, as no dependence can be placed upon the presence or absence of a particular hue. It is the face of one seriously ill with some form of sickness. No doubt, in many instances, there is a greyish aspect, but in others, except the pallor, there is little observable change. The greyish earthy hue is much more frequently seen in cases of infective thrombosis. The yellowish hue is indicative of pyæmia.

The face, however, is expressionless, passive, and cloudy. It may assume a meaningless smile, with which the features are not lit: it is too mechanical.

An *odour* of a putrid character, from the breath of a patient suffering from cerebral abscess arising from infective otitis media, is common in this period of the disease. The tongue is generally thickly furred, and if the teeth are not cleaned they become covered with sordes. The odour from the breath might be attributed to the state of the mouth and to disordered digestion were it not for the fact of its close resemblance to, or absolute identity with, that emanating from the middle ear. The character of the odour persists, though it is lessened in degree, after the mouth and ear have been cleansed. Occasionally the odour is so marked that it is perceived on approaching the patient's bed. At other times it is only discerned in close proximity to the mouth of patient. The patient complains, at the same time, of a nasty taste in his mouth, and also perceives a bad odour.[1]

It must be borne in mind that a similar odour is perceived in neglected cases of otitis media without intracranial complications. The putrid odour from the breath is often much greater in infective thrombosis which has proceeded to purulent infective pulmonary infarction. The odour of the expectoration is then of the same character.

Rigors are unusual during the second stage. They are seldom repeated after the initial period. They may, however, recur later if fresh foci occur round the periphery of the abscess, or if the abscess leaks into the ventricles, or into the subdural cavity, provided death does not ensue too rapidly thereafter.

The infrequency of rigors in cerebral abscess is in marked contrast to their persistent recurrence in infective thrombosis of the sigmoid sinus and its sequelæ, in which, throughout the whole course of the disease, rigors occur every few days, followed by the characteristic perspiration, and they increase toward the fatal end.

Emaciation is frequently observed toward the latter portion of the second stage, and, when accompanied by low temperature and constipation, it is of value in diagnosing cerebral abscess. On the other hand, when it appears after great pyrexia, repeated rigors, followed by sweats and diarrhœa, it is then due to infective absorption, probably from infective thrombosis of the sigmoid sinus, and not to uncomplicated cerebral abscess.

[1] The disagreeable taste in the mouth and the bad odour perceived by the patient cannot be attributed to perverted sensation originating in the temporo-sphenoidal lobe, as there is the patent fact of the presence of this putrid pus in the ear and the expectoration has a putrid odour.

The *reflexes*, superficial and deep, are sometimes affected in cerebral abscess, but hitherto they have seemed to be so uncertain that little dependence can be placed on the variations which they present.

Muscular fibrillæ are often seen toward the end of the second stage, and are marked in the terminal period. Myoidema is also present in many instances about the same time.

These are in no way diagnostic of cerebral abscess, as they are found in cases of serious illness of a wasting kind and of exhaustion from numerous causes.

Optic neuritis is frequent in cerebral abscess, especially in the later periods of the disease—toward the end of the second stage—and in those abscesses which have attained a considerable size. Cerebral abscess being an acute affection, and papillitis seldom commencing at the outset of the disease, the neuritis does not as a rule attain great intensity, as the time is generally too short for its complete development. It is seldom seen to such a marked extent as in cases of tumour of the brain. The optic neuritis seen in cerebral abscess is at an early stage of its development, is still on the increase, and is advancing at a rapid rate. The papillitis persists and may even become intensified for days and weeks after the evacuation of the abscess. Thereafter it may remain in a stationary condition for many weeks. Subsequently, in cases where the neuritis has reached an advanced stage, the absorption is slow, and traces of it are distinct at the end of six or eight months. It is rarely followed by neuritic atrophy. As in other intracranial affections, it is seldom found with equal intensity in both eyes, being generally marked in one, while the other is only slightly affected. In abscess, the neuritis is not always more intense on the same side as the lesion. Sometimes it is the reverse, even when the abscess is situated in the middle and rather to the posterior part of the temporo-sphenoidal lobe. The eyesight is seldom much affected from the degree of papillitis present in abscess. Optic neuritis has been found in abscesses of the frontal and temporo-sphenoidal lobes, and in those of the cerebellum. Optic neuritis will be found neither in cases of cerebral abscess which run a very rapid course, the period necessary for its production being too short, nor where the cerebral abscess is small and the degree of surrounding inflammatory action is slight. Probably in some instances where papillitis is present in abscess, it is caused by the accompanying meningitis,

but in others there is no evidence of meningitis, all the symptoms pointing to simple cerebral abscess. In the former the optic neuritis is probably due to irritation extending along the nerve or its sheath.

Optic neuritis may arise from quite a number of intracranial affections—meningitis, tumour of the brain, and cerebritis. It has been said[1] that it may even be produced from suppuration of the tympanic cavity, without evidence being present of any intracranial affection, in which case it is supposed to arise through damage to the carotid plexus of the sympathetic inducing vasomotor disturbance in the optic nerve and fundus independent of any brain disease. In any case where it might be supposed to exist without intracranial disturbance it would be difficult to prove without post-mortem evidence that it was not due to a slight degree of meningitis. Infective disease of the middle ear with erosion of the tegmen tympani is so often accompanied by a slight degree of meningitis, sufficient to produce slight neuritis, which would persist for months after recovery from the meningeal attack, that the papillitis in such rare cases as are referred to above might be accounted for in this way. Besides, if the irritation in the tympanic cavity were sufficient to produce an effect upon the carotid plexus of the sympathetic, it might also induce irritation in the perivascular sheaths of the carotid venous plexus, as well as in those veins which pass directly from the tympanic cavity into the membranes. Further, it must not be forgotten that some systemic diseases, such as albuminuria, are often accompanied by optic neuritis.

The absence of optic neuritis does not exclude cerebral abscess; but when it is present and presents the special characters already described, it is, when taken along with the other symptoms, a distinct aid to diagnosis.

The *aural examination* is of great importance in diagnosis. A chronic infective otorrhœa, which usually, though not always, smells foully, and perforation of the membrana tympani, with granulation tissue protruding, are the local conditions most favourable for the development of intracranial inflammations. If the roof of the middle ear can be seen, it is possible that erosions of the tegmen may be visible. In one case of abscess of the brain related by Dr. Barr, the auriscope showed a drop

[1] Mr. Barker, *Hunterian Lectures on Intracranial Inflammation starting in Temporal Bone.* 1889.

of pus accumulating every few minutes on the roof of the tympanum, evidently emanating from a minute perforation in that thin partition. That was proof of intracranial suppuration, but it is very rarely that distinct evidence of this kind exists. The tympanic cavity is generally blocked with granulation tissue which is afterwards found to extend into the mastoid antrum. By the use of a probe, rendered aseptic, introduced into the middle ear, and directed to its roof, erosions have often been detected, and several times perforations of the tegmen have been discovered.

Swelling and *redness* over the *mastoid process* seldom accompany cerebral abscess in adults, and, when present, no reliance can be placed on them as indicating intracranial suppuration. In many instances of intracranial suppuration, the mastoids are thickened and eburnated, and therefore are likely to prevent the formation of the external appearances so common in more superficial inflammations. Tenderness on percussion over the mastoid is frequent in cerebral abscess, but this is more pronounced in phlebitis and infective thrombosis of the sigmoid sinus, as is also the tenderness on pressure over the jugular. After exposing the mastoid antrum and the tympanic cavity from behind the auricle, further evidence of erosion of the base of the skull, toward the cerebellum and temporo-sphenoidal lobe, may be discovered.

TERMINAL STAGE.—The disease, when allowed to pursue its course uninterruptedly, ends as a rule in death. It does so in at least two ways: by gradually deepening stupor and coma, and by a train of symptoms brought about by oozing of the pus or complete bursting of the abscess on to the surface of the brain or into the ventricles.

When the abscess leaks on to the surface of the brain, or into the ventricles, fresh irritation is established, and the inflammatory action spreads with great rapidity. In some instances, without actual bursting, the inflammatory zone surrounding the abscess may spread in such a way as to involve in acute inflammation the pia mater, and set up acute leptomeningitis. This is generally heralded by a new train of symptoms—vomiting, restlessness, temporary squinting, flushing, and erratic rigidity of the limbs, clonic spasms, trepidation, and prostration, the pulse meanwhile becoming quick, the breathing hurried, and the temperature high.

When the abscess bursts into the ventricles, the symptoms are much more sudden and severe. The whole aspect of the patient changes with marked rapidity. The pupils become widely dilated, the face livid, the respiration greatly hurried, and either shallow or stertorous. The temperature rises within a few hours with a bound from subnormal to 104° F. or 105° F.; the pulse from 40 or 50 per minute quickly reaches 120, and over. There are muscular twitches all over the body, possibly associated with convulsions and tetanic seizures (opisthotonos and emprosthotonos), and these are followed by coma and speedy death. This stage may terminate within six or twelve hours, from the first indication of bursting into the ventricles till death ensues.

Convulsions, coma, and death are well illustrated in Case XXXIV., an abscess in the frontal lobe. The phenomena attending the bursting of the abscess into the ventricles are marked in Case XXX. In another case (XXXIII.) of abscess in the frontal lobes, the symptoms point to oozing of pus into the ventricles, and in Case XXIX. there were besides the above symptoms, slight indications of spinal involvement.

Apparent natural cure.—No doubt abscesses, once they have become encapsulated, may remain at rest for long periods, during which the patient is enabled to go about and perform work without presenting any obtrusive symptoms. This is illustrated in Case XXX., where the patient had an encapsulated abscess evidently of long standing, and while affected with the abscess she was able to perform her work as a domestic servant, though her mistress thought she was sometimes stupid, especially if spoken to sharply. In this instance the patient died from the formation of a very large abscess round the periphery of an old one. It is also quite possible, when the abscess has become encysted and the patient is in good health, that such an abscess may not only diminish in bulk, but become finally absorbed. It need scarcely be said that no positive proof of such an occurrence exists. The capsule of the abscess is much more likely to become thickened and remain as a foreign body. The capsule and contents may even become calcified. Gowers mentions that death has occurred from an abscess after a latent period of twenty years.

Cerebral and cerebellar abscess may find an exit through the skull, and may be poured out externally. Cerebral abscess in the temporal lobe may find an outlet through softened membranes

and eroded tegmen tympani. Such cases are narrated in the clinical records annexed, as well as a case of cerebellar abscess where a sinus led through a carious erosion in the sigmoid groove into the abscess cavity, the venous sinus having been obliterated. It is possible that such abscesses may drain in this manner, after which recovery may take place. At the same time the exit for the pus is often by a very tortuous path, and the exit aperture so fine, and generally occluded with granulation tissue, that the evacuation of the abscess is incomplete. Besides, it is always a source of danger, as fresh pathogenic organisms may enter from without, and set up renewed disturbance in the brain or cerebellum.

Localizing symptoms.—In addition to symptoms which indicate the presence of abscess in the brain there may be others of a different kind which show the particular area in the brain which is the seat of the abscess. These are termed localizing symptoms and, when present, are of great value.

Localizing symptoms are only occasionally found in cerebral abscess, because in the majority of instances it is situated outside of the motor area. The chief seats of abscess arising from infective otitis media are the temporo-sphenoidal lobe and the cerebellum, and from infective injury about the forehead the frontal lobe is frequently affected. In any of these regions an abscess may form without exhibiting localizing phenomena, and this is especially so if it be of small size. When large there is frequently evidence of a more or less directly localizing character, even when the abscess is situated in the temporo-sphenoidal lobe.

Pupillary phenomena are not infrequently manifest in abscess of the brain. When the abscess is seated in the temporo-sphenoidal or frontal lobe, the pupil on the same side as the abscess may either become myotic or mydriatic, accompanied by a degree of stability. When the abscess is small and produces cerebral irritation, the pupil on the affected side may be more or less contracted and sluggish. When the abscess is large and exercises considerable pressure, the pupil on the affected side is prone to stabile mydriasis. An abscess which is small, and produces a stabile myosis to begin with, may, as it augments in bulk, produce a stabile mydriasis in the same eye. Occasionally the only difference detectable is sluggishness of one pupil to both light and accommodation while its neighbour acts freely. In a case of cerebral abscess, a sluggish pupil with either mydriasis or myosis on

the same side as an infective injury to the skull, or a unilateral otitis media, affords further evidence of the abscess being on the corresponding side of the brain.

General cerebral meningitis, as a rule, affects both pupils equally, and they usually are then contracted until the pressure symptoms at a late stage supervene, when they both become dilated and fixed. Infective thrombosis of the sinuses, with the exception of thrombosis of the cavernous sinus, rarely affects the pupils, and then the proptosis, the stabile mydriasis, and more or less complete blindness, at first on the same side, then implicating the other eye, make the distinction easy.

Elicitation of a differential cranial percussion note.—The elicitation of a differential cranial percussion note as an aid to cerebral diagnosis in certain gross changes of the intracranial contents, especially in children, has been practised by the author as opportunity offered during the last ten years, and as it has been found to be useful in diagnosis the following may assist those desirous of practising it.

The percussion note is obtained by the cranial walls vibrating when struck, the note being modified by the consistency and volume of the contents and their relative position to the bone. The sound elicited depends first on the susceptibility of the skull to vibrate, and secondly on the effect which the intracranial contents exert on these vibrations. When struck, a thin cranium vibrates more easily than a thick one. A skull may be so thick that it vibrates little, if at all, to ordinary digital percussion. In the cranium of an infant, whose bones are only united by a membrane, and where they lie somewhat loosely on the brain, the percussion note elicited is so slight, dull, and flat, as scarcely to be perceptible. Should, however, the contents of such a cranium increase sufficiently to produce tension of the whole parietes—bones and intervening membrane—then the note becomes clear, particularly as the density of the contents of such a case is low—the brain containing a considerable quantity of serous fluid. In the child whose fontanelles remain open, especially in delayed closure from serous over-distension, a clear drum-like note is elicited on percussion. A comparatively thin skull may emit almost the same kind of note as a thicker one when the contents of the cranium are intimately applied to the whole of its surface. Where the ordinary density, consistence, and relation of healthy

brains to the cranial bones exist, a dull note is produced on percussion, but where the contents are more fluid the bones will vibrate more readily, and a clearer percussion note will be obtained. It is almost superfluous to note that the amount of hair on the head and the thickness of the scalp must exert a modifying influence on the percussion note, which is best elicited after shaving the head.

Method of eliciting the note.—This may be done by the ordinary instruments used in percussing the chest or by the tip of the middle finger striking lightly on the cranium. The latter is safer, and answers all ordinary requirements. The note is best elicited near the pterion or a little posterior to that point, but when distinct it may be produced all over the parietal and frontal regions.

By placing the ear of the investigator in contact with the head of the patient the percussion note conveyed is heard much louder and, in some instances, its quality may be more easily estimated, though, on the other hand, contact with the head of the investigator may limit the vibrations and somewhat deaden the sound. If possible, the percussion ought to be practised when the patient is in the upright position, as when the head rests upon a hollow pillow, or even upon a feather pillow, the sound is apt to be altered by the contact.

The percussion of the cranium of healthy children imparts a dull sound which is nearly equal all over the vault. In some, who are either healthy or at least have no obtrusive diseased condition, there is a slightly more resonant note elicitable over the pterion or a little way behind it, but the degree of resonance is only slightly clearer than normal. In healthy adult heads the note is generally high pitched, imparting the idea of solidity, most pronounced along the base or over the occipital protuberance. In many adults the bones of the cranium are too thick and dense to permit of active vibrations on ordinary digital percussion.

In some children who have the typical enlarged cranium and small face so often associated with infantile rickets, the percussion note is more resonant than normal, but as this alteration in note does not obtain in all such cases, it must be dependent upon a variable factor. In some cases of rickets when the clear percussion note was obtained, it was afterwards proven that the lateral ventricles were distended with fluid which in these cases

probably gave rise to the peculiarity of the note. In others the porosity of the bone of the rachitic skull seemed the only cause.

The percussion note in over-distended lateral ventricles.—When the lateral ventricles are distended with serous fluid, as may be occasioned by cerebellar tumours pressing on the fourth ventricle, or by occlusion of the veins of Galen, or otherwise, the percussion note is markedly altered, the resonance being greatly increased. Besides the increased resonance, there is an important feature which may be demonstrated. The percussion note elicited at a given spot on the cranium, such as the pterion, varies according to the position of the head. While the person sits with the head upright, the most resonant note is brought out by percussion toward the basal level of the frontal bone and squamous portion of the parietal. If the patient hangs his head to one side, so that one parietal is placed fairly below the other, the greater resonance is found on percussion of the lower parietal. Reverse the position, and the same note is elicited on the opposite side of the head, which is now the lower, the greatest resonance being found at that part of the skull nearest the lateral ventricles, and which for the time is at the lowest level. These observations tend to indicate that the quality of this note is not dependent upon the mere density or diameter of the cranium, but to a large extent upon the consistence and arrangement of the intracranial contents relatively to the osseous walls. The exact musical quality of the note is difficult to describe, but when heard, it conveys the idea of hollowness. One such case in which the above phenomena were clearly marked was observed to a conclusion. The percussion note was not so clear at first as it ultimately became, the resonance increasing as the disease advanced. Post-mortem cranial transverse frozen sections were made, so as to secure the relative position of the intracranial fluid and brain to the skull. These showed that there was a large tumour in the cerebellum which pushed the right lobe to one side, and blocked the fourth ventricle. There was great distension of the lateral ventricles with fluid, with consequent thinning of the substance of the cerebrum, and reduced amount of subarachnoid fluid. Since then, ten cases have been similarly observed to a conclusion, and in all of them the post-mortem showed over-distended ventricles, the brain tissues being thinned.

Many similar observations abundantly verify the foregoing statement. The author has found this clear percussion note in over forty children and young adolescents who have had distended ventricles arising from many different causes. In tumours of the cerebellum it is an aid to diagnosis. When present, along with abscess, it points to involvement of the cerebellar fossa. In the annexed clinical record instances are given in which cerebellar abscess was associated with enlarged ventricles, the percussion note being more highly resonant than usual. On several occasions in examining children and young adolescents whose symptoms were obscure, but pointed toward cerebellar tumour, the typical cranial note described above greatly aided the diagnosis, the correctness of which was ultimately fully verified.

The elicitation of the percussion note in most cases does not seem to incommode the patient. In many instances the children were amused by it, and in several the patients were percussed while asleep.

The cracked pot sound elicited on percussion of the head in extensive fractures of the skull, when large areas of bone are detached by fissures, the author has only been able to detect on three occasions in the adult. In one who had a very extensive fracture of the parietal bone with some depression, from which ran a fissured fracture toward the forehead, while another travelled toward the base (there were basal symptoms), the percussion note elicited over the shaven head was marked and characteristic.

The facts here adduced indicate that a field, though a limited one, exists in which the elicitation of a differential percussion note is of diagnostic service when carefully considered along with other data. The obscurity of cerebellar lesions at an early stage, and especially in children, is such that every point which assists in their detection is of value.

"*The latent period*" in abscess formation is a phrase often used in text-books, and by writers on cerebral abscess. It is difficult to understand to what this precisely refers, and it is apparently used in various senses by different writers. If it be applied to an acute abscess, it can only be to the second period described, in which there is a lowering of the temperature, slowing of the pulse, and a general blunting of all the senses. It will be readily seen, however, from the description previously

given of this "second stage," that it abounds in symptoms of cerebral disturbance, which, taken collectively, point strongly to encephalitis or abscess. Therefore the phrase, "latent period," is inapplicable, though it is true that in many cases the symptoms may be so unobtrusive that they may be overlooked unless specially sought for.

If, on the other hand, the phrase be used to indicate a period in those much rarer cases—chronic encapsulated abscess—which have remained quiescent for years, surrounded by a layer of connective tissue, then it is quite appropriate, as the symptoms may disappear along with the subsidence of the encephalitis surrounding the capsule, and only reappear when fresh encephalitis supervenes in the periphery. The abscess may then comport itself as a foreign body, or as a tumour whose growth has ceased, at least for the time. There may be a true period of latency in such cases which may last months or years, or, if absorption of the pus occur, may continue permanently; or the latent period may be broken if the wall burst, and the contents suddenly escape on to the brain surface or into the ventricles, when symptoms of an aggravated kind may suddenly occur somewhat resembling apoplexy (see under heading, "Symptoms," Bursting of Abscess into Ventricles, p. 144).

Evidence of intracranial abscess obtainable from the quantity of pus issuing from the external auditory meatus.—The quantity of pus issuing from the middle ear by the external auditory meatus ought always to be noted, with the view of ascertaining whether it be greater than what would be likely to emanate from the middle ear and its recesses. If it be distinctly greater, there must be a cavity within the cranium from which this pus flows. In several instances in the author's practice the amount of pus which issued in twenty-four hours from the external ear was greater than could emanate from the middle ear and its recesses; this fact, therefore, raised the presumption of intracranial abscess. In one case also where incisions had previously been made over the mastoid process by another surgeon, the amount of discharge issuing from the sinuses when seen by the author was so great as to indicate the probability of intracranial abscess.

Evidence obtained by seeing pus issuing from an erosion in the tegmen tympani.—Careful auriscopic examination occasionally may reveal erosions in the tegmen through which pus issues in drops, which would be a clear indication of intracranial abscess (see p. 142).

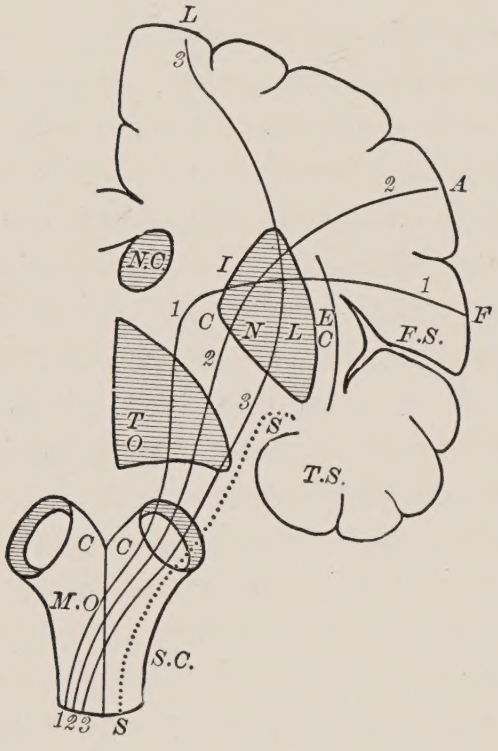
Symptoms of empyema of the frontal sinuses.—The patient complains of a dull heavy pain or sensation in the front of the brow sometimes described as headache. It is more annoying from the sensation of dulness than of acute pain. Percussion over the sinuses usually increases the pain. The patient feels easiest in the upright position; while stooping or the prone position increases the aching. Purulent secretion from the nose is frequently free, and occasionally it is abundant from the inner angle of the upper eyelid. When the abscess bursts into the orbital cavity diplopia frequently follows. When the bone is opened over the frontal sinus especially near the middle line, the mucous membrane if in a physiological condition is of a dark-red or livid hue, and expands and contracts with the respirations.

LOCALIZING SYMPTOMS.—*Localizing symptoms of abscess in temporo-sphenoidal lobe.*—Abscess of the temporo-sphenoidal lobe when small presents no localizing symptoms, though the pain at the outset generally originates in the ear on the same side as that on which the abscess is situated. When large, the adjacent cerebral areas and nerves are apt to be involved either by participating in the extension of the inflammatory action surrounding the abscess, or by pressure more or less directly applied. Of the adjacent cerebral structures whose function is ascertained, those most apt to be affected are—first, the lower portion of the ascending frontal and parietal convolutions; second, occasionally the base of the posterior part of the third frontal convolution along with the adjacent part of the ascending frontal; third, in connection with the first and second, it must not be forgotten that the posterior half of the first temporal convolution may also be affected: the last two are especially apt to show evidence of being implicated when abscess is on the left side of the brain. Possibly the motor and sensory strands in the internal capsule may be pressed on. Of the nerves, the third is most frequently implicated.

Hemiplegia on the side opposite the lesion is occasionally found in large cerebral abscess of the temporo-sphenoidal lobe, and a degree of paresis of the opposite limbs is probably present more often than is noted, especially where instruments of precision are not used in measuring the relative grasp of the hands. In certain cases, the hemiplegia is evidently due to pressure, as the restoration of function follows soon after the evacuation of the pus, an improvement in the paralysis being observable within a few hours thereafter, and complete restoration within a few days. In

others, the parts concerned have evidently participated in the extension of the inflammatory action, as the paralysis may remain stationary for days after the evacuation, while traces of it have been detectable weeks subsequently. It is important to ascertain the precise order in which the various parts became implicated, as it may assist in determining whether the motor cortical centres or the strands of the internal capsule have become involved. When the face becomes first affected, the arm second, and the leg

FIG. 50.—SCHEMA ILLUSTRATING SEQUENCE OF PARALYSIS CAUSED BY ABSCESS IN TEMPORO-SPHENOIDAL LOBE.



F, Facial. *A*, Brachial. *L*, Crural-motor areas. 1 1 1, Facial, 2 2 2, Brachial, 3 3 3, Crural motor strands from cerebral cortex through *I.C*, internal capsule, and *C.C*, crus cerebri, to opposite side of spinal cord, *S.C*. *S.S*, Sensory tract. *T.O*, Optic thalamus. *N.L*, Lenticular nucleus. *N.C*, Caudate nucleus. *E.C*, External capsule. *M.O*, Medulla oblongata. *T.S*, Temporo-sphenoidal lobe.

slightly, if at all implicated, especially when the sensation in these parts has been preserved, the abscess in the temporo-sphenoidal lobe is, in all probability, exerting an influence upon the cortex of the ascending convolutions from below upwards (see Diagram 50). If, however, leg—arm—face was the relative order of the implication, or if they were simultaneously affected, and especially if there was a coincident sensory paralysis, then the probability of secondary involvement of the internal capsule would be greater. (There are no cases recorded, however, in which there is un-

doubted evidence of secondary involvement of the internal capsule from abscess in the temporo-sphenoidal lobe.) There is doubtless difficulty in obtaining a clear history as to those points unless the patient has been under the scrutiny of trained observers, but occasionally the friends are able to supply positive information on that score.

It must be borne in mind that paralysis of the facial nerve is common in advanced destructive lesions of the mastoid antrum and tympanum, and hence is seen in many cases of abscess in the temporo-sphenoidal lobe. The paralysis in this case would be on the same side as the lesion. To distinguish a paralysis of the facial muscles, produced by a cortical lesion, from one produced by paralysis of the facial nerve, is necessary in some of those instances where the side on which the lesion is situated is as yet undetermined. In that produced by a cortical lesion, the paralysis is seldom so intense: the patient can usually shut the eyelid, and there is a slight manifestation of the power of emotional expression on the affected side, and the sense of taste in the anterior two-thirds of the tongue remains *intact.* These are absent when there is complete peripheral paralysis of the facial nerve.

Aphasia has several times been noted as accompanying abscess in the left temporo-sphenoidal lobe. The special form which it has assumed has not in most cases been specified. Close inquiry has shown that in some instances, at least, the term has been loosely applied. The general mental obscuration so frequently present in cerebral abscess is such as to render the detection of aphasia difficult. It is of diagnostic importance to differentiate the variety of aphasia. If it be sensory aphasia—word deafness —the posterior half of the first temporal convolution would be involved, hence the abscess would probably be situated toward the posterior aspect of the temporo-sphenoidal lobe; whereas, if the aphasia be motor, the bases of the third and ascending frontal convolutions, or the conducting paths from the cortex, would be the seat of the implication. One case of aphasia in connection with temporal lobe abscess is reported by Drs. Greenfield and Caird.

There has been no case of temporo-sphenoidal abscess under the author's care in which distinct motor aphasia has been a feature, though in one instance it was noted by the medical attendant as having existed before the patient was brought under the author's observation. Of sensory aphasia, however, Case

No. XXXVII. is a marked example in which the posterior aspect of the first temporal was involved.

Paralysis of *the third nerve* on the same side as the lesion is to be looked for, as it occurs in some cases of large abscess in the temporo-sphenoidal lobe. When the paralysis is complete, the ptosis, the external strabismus, and the fixed and dilated pupil are characteristic, the paralysis of all the external ocular muscles, except the superior oblique and external rectus, preventing the eyeball from moving, except outwards and slightly downwards. Sections of the frozen head show well the relations of the third nerve to the temporal lobe, and how distension of this lobe may exercise pressure upon the third nerve.[1] The third may no doubt be affected by extension of meningeal inflammation, while the fifth may escape for a time as it is sheathed in the two layers of the dura mater; on the other hand, the rapidity with which the paralysis disappears from the muscles innervated by the third, after removal of the pus, in the majority of instances renders it probable that pressure, and not inflammation of the meninges, is the cause.

If one finds paralysis of the third on the same side as the lesion, paralysis of the opposite side of the body, commencing in the face, answering the tests of paralysis originating in the motor cortex and remaining most marked in the facial muscles, then involving the brachial muscles to a less extent, the lower limb remaining free, and all these being present without loss of sensation, the great probability is that the lesion is a large one situated in the temporal lobe. The Case No. XXV. confirms this point, as does also the second abscess in the list, and many others elsewhere reported bear it out. The last case of large abscess in the temporo-sphenoidal lobe, Case No. XXVI., likewise had this feature to a marked extent.

In one case of abscess of the temporal lobe (No. XXV.) the patient was clearly in a *somnambulistic* state from a period before he left his own home till he found himself in bed in the Royal Infirmary, fully twelve hours after the operation—in all at least thirty-six hours. Though he seemed awake, and answered questions intelligently and correctly during that period, yet when he awoke, twelve hours subsequently to the operation, he had no recollection of anything which had occurred meanwhile. It may be a somewhat similar condition which Dr. Hughlings Jackson

[1] See Atlas of Head Sections, Plates 41 and 45.

has termed a "dream state," and which he has found accompanying a tumour in one of the temporal convolutions.

Word deafness and psychical blindness.—In an abscess which involved the posterior third of the first temporal convolution, along with the adjacent angular gyrus (Case No. XXXVII.) word deafness and psychical blindness were both present.

CASE XXV. *Case of cerebral abscess, due to infective otitis media, diagnosed to be of large size, and situated in the temporo-sphenoidal lobe. Points of diagnosis. Operation. Recovery. Coming under observation on the twenty-first day of his illness. Duration of illness and convalescence six weeks.*

G. S., aged 21, admitted to Royal Infirmary, 14th May, 1888, affected with symptoms which had been certified as meningitis, but which were obviously those of cerebral abscess, situated in the temporo-sphenoidal lobe.

History.—*Heredity.*—Patient's parents were both dead; the cause of death could not be ascertained. He belonged to a family of seven, of whom two—a brother and a sister—were dead; one having died from phthisis, the cause of death of the other being unknown. Two of the brothers who were living, and also the sister who died, were deaf and dumb, all of them having had purulent discharges from the ears. *Personal.*—Patient had a purulent discharge from his left ear since childhood, but with this exception had been in good health, and able to pursue his work as a blacksmith regularly, until three weeks ago. Five months ago he had a fall, which caused a wound on the top of the head, which soon healed and did not prevent him from working.

Present Illness.—Three weeks ago he complained of headache, confined to the forehead and temples on both sides, of such a severe character as to cause him to desist from work. Soon after its onset he became sick and vomited. Several times during the second week he had feelings of cold, and on three occasions these amounted to shiverings (rigors), the last on the evening of the 13th instant. Meantime he was getting drowsy, wakening only when roused, and tossing about in bed. On the 11th May his speech became thick, and next day the upper left eyelid was seen to droop. His friends stated that he had rapidly emaciated since his illness. There were no convulsions.

State on Admission.—*Aspect.*—Patient had a drowsy dreamy look; his face had an ash grey colour tending to lividity. He lay in bed with his left eyelid closed, while his right was only partially shut, but when roused he opened his right eyelid widely and answered questions slowly but intelligently, though his answers were laconic. There was a very offensive odour perceived when approaching his bed, coming especially from his breath. His teeth and tongue were covered with sordes.

Motor Functions.—*Eyes.*—There was ptosis of left eyelid, the left pupil was in a state of stabile mydriasis; there was a marked degree of paralysis of all the ocular muscles, with the exception of the external rectus, which was

unaffected. There was occasionally a barely perceptible external squint. The right pupil was normal, and the right eyeball moved freely in all directions.

Facial paralysis from brain lesion.—The right side of the face was paralysed, though the emotional expression was retained to a slight extent, as might be seen when he attempted to smile. Though the right eyelid remained partially open while asleep, he could close it when he made an effort. The tongue could be protruded in a straight line when his attention was maintained. It was not distinctly thicker on the one side than on the other. When he lay on the right side in bed the saliva trickled from the mouth.

Right Arm.—The right arm was partially paralysed. He could lift it to his head by a feeble movement, but he was only able to press the dynamometer to ten, and once to fifteen, while with the left the pressure indicated forty-five kilos. A tendency to wrist-drop was present in the right arm. The posture sense was retained.

Leg.—He walked with a staggering gait, but there was no difference between the muscular and sensory power of the individual lower limbs.

Reflexes.—The cutaneous reflexes were normal. Patellar reflex possibly a little exaggerated ; no ankle clonus. There were constant muscular tremors over the body generally, affecting the individual muscular bundles, especially marked in the pectorals.

Sensory Functions and Special Senses.—There was no impairment of sensation or of special sense with the exception of a diminished power of hearing on left side, and a slight impairment of sight, which, however, was difficult to test owing to his mental state. Taste and smell seemed acute.

Ear.—The left ear was found to contain a small quantity of fœtid pus. The only vestige of the membrana tympani was a fringe of granulation tissue, and at the upper aspect of the roof of middle ear a minute quantity of pus appeared. The probe cautiously introduced detected bare bone in roof of middle ear. The hearing was very dull on left side, though bone conduction was good. On right side hearing was normal.

Eye.—Ophthalmoscopic examinations showed that the left retinal vessels were to a marked extent engorged and tortuous and partly veiled by exudation, while those of the right were affected in a similar manner, but to a less degree.

Pulse was markedly cerebral in character, rather feeble, from forty to fifty per minute, occasionally intermittent. The temperature was from 98° to 99° F. Respiration rather slow, and occasionally sighing.

Fixed Pain. Temporal Region.—At the outset of his illness he complained of severe frontal headache, which extended to both sides of the head equally ; when in the ward he complained of a fixed pain over the left side of head, which was increased on movement. While percussion over the head generally was borne without complaint, he winced and strongly objected to percussion over this area.

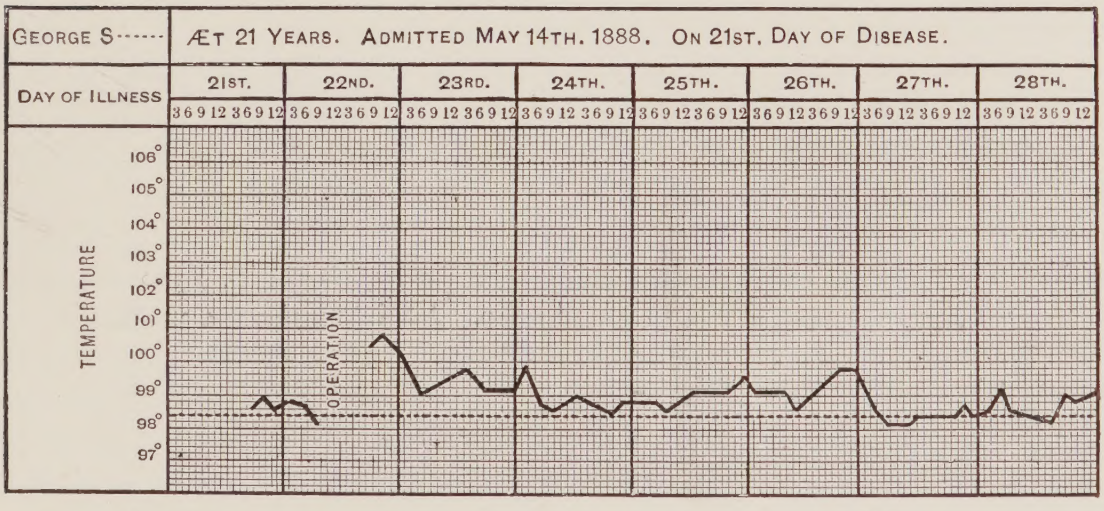
The note elicited on percussion was thought to be more hollow over the left temporal region than elsewhere, but this could not be distinctly determined as he did not remain sufficiently quiet during the investigation. When

pressure was made over the left internal jugular, both in front and behind the sterno-mastoid, patient winced.

General Mental Condition.—The most marked feature about his mental state was his drowsy condition. Were he left alone he would doze on, only occasionally moaning from the pain in his head. When roused he answered questions intelligently and correctly, but he had a marked want of sustained attention, and was apt to drop asleep before the answer was quite finished. As a rule, his answers were laconic, excepting when he complained of the pain in the head, or stated that he felt cold, when he heaped the blankets round him. He never volunteered a statement. He never asked for anything to eat or drink, but drank when the vessel was held to his mouth. There was no amnesia.

Urine.—He had passed no urine for some time, and could not pass water voluntarily. Twenty-seven ounces were withdrawn by catheter. Its specific gravity was 1030; it contained neither albumen nor sugar. Twelve hours after, eleven ounces were drawn off.

CHART F.—ABSCESS OF BRAIN.



The temperature chart of a typical uncomplicated case of cerebral abscess.

Bowels.—Obstinate constipation. A purgative and several enemata failed to move bowels.

Diagnosis of Lesion.—From the sudden onset and rapid course of the illness, occurring in a person the subject of chronic fœtid otorrhœa due to otitis media, ushered in by severe headache and slight vomiting, followed after the lapse of some time by sensations of cold, several slight rigors, drowsiness and mental disturbance, rapid emaciation, low temperature, slow pulse, and optic neuritis, cerebral abscess was diagnosticated.

Locality.—The paralysis of the oculo-motor on the left side, though involving the whole thickness of the nerve, was yet not absolute in degree; the engorgement of the retinal vessels and tortuous veins was most marked on the left fundus; the paralysis of the voluntary power of the right facial muscles, though the emotional expression was partially retained; the ability

to close the right eyelid, the paresis of the right arm and the drop-wrist, the absence of any sensory derangement, and the retention of the posture sense, pointed to a large abscess in the left hemisphere, affecting at once the left third nerve and the lower and middle portions of the central convolutions.

It was obvious that such a large abscess could not be situated in the central convolutions without causing, besides the probable involvement of the crural centre, such an absolute paralysis of the muscles of the arm and face as did not exist.

The order of succession of the paralyses, and the degree in which the various parts were implicated, were also important—the face being the first paralysed, then the arm, the voluntary facial muscles being markedly affected, and the slighter implication of the brachial muscles, which however were becoming more deeply involved, even during the few hours he remained under observation prior to operation. Motor changes, both as regards their sequence and the degree in which they affected individual parts, pointed to the conclusion that there was an involvement of the function of the central convolutions from below upward, such as the writer has previously found in cases of large abscess situated in the temporo-sphenoidal lobe. The paralyses was produced by indirect pressure, or by extension of the inflammatory action from contiguity of tissue, or by both.

The absence of sensory impairment showed that Charcot's crossway was not involved, therefore the abscess must have been situated considerably outside of the internal capsule. An abscess which was clear of the internal capsule, which affected at once the third nerve and the lower portion of the central convolutions—the latter becoming implicated from below upwards—and while doing so did not *directly* involve either, would be situated in all probability in the temporo-sphenoidal lobe. Had the abscess pressed upon the crus cerebri and so produced the paralysis, the parts would have been affected in the inverse order, the leg first, the face last, whereas here the face was first and the leg was not involved. (See Schema, page 152.)

May 14th, 1888.—Dr. Reid was afterwards asked to make an independent ophthalmic examination. The following is his note:—

Ophthalmic Examination. Left Fundus.—"There is well-marked papillitis, the centre of the disc presenting about – 6 D.'s of refraction, whilst the region of the macula presents about – 4 D.'s of refraction.

The outline of the disc is veiled by exudation, which seems to extend some distance beyond its margin.

The retinal vessels, the veins especially, are congested, tortuous, and at points veiled by exudation. Minute hæmorrhages of a streaky form are present near the upper and outer margin of the disc.

Conclusion.—The appearances present point to inflammatory exudation, without any distinct evidence of being associated with *great* pressure.

Right Fundus.—The appearances of the right eye are similar to those of left, but to a less degree both as regards prominence of the disc and tortuosity of vessels. There is the same veiled appearance of the outline of the disc."

It was evident that operation was imperative.

As the patient had come under observation late at night it was midnight before the examination was completed, the operation was therefore ordered in the early morning. Meanwhile he was placed under the charge of a special nurse. The head had to be shaved and the scalp rendered aseptic.

In the morning the paralysis of the right arm was more pronounced, and the drop-wrist absolute, the drowsiness greater, the pulse forty per minute, and the left pupil was in a state of absolute stabile mydriasis.

Operation (May 15th, 1888).—Patient was anæsthetized by Dr. Fox. The middle ear was cleansed, the granulation tissue removed, and the parts rendered aseptic. The mastoid cells were quickly exposed and washed out. There was very little pus found in them. The bone of tegmen was, however, bare at one spot, and covered by a small quantity of pus. Afterwards a vertical wound was made into the scalp, exposing the bone. The soft parts were then dusted thickly with iodoform. A half-inch disc of bone was removed from the squamous portion of temporal, at a point an inch above the centre of the external auditory meatus.

The dura mater was normal. It bulged slightly into the aperture, and there were no cerebral pulsations visible. It was incised, when the cerebral tissue, which had been firmly pressed against the dura mater, protruded into the osseous gap. There was no cerebro-spinal fluid in the subdural or subarachnoid spaces, which, indeed, seemed to have no existence as spaces, as the pressure had obliterated them. The exposed cerebral tissue was normal in aspect, but did not pulsate. A hollow aseptic needle was introduced into the brain in a direction which, if it penetrated far enough, would strike the petrous portion of the temporal. It had penetrated three-quarters of an inch when it was felt to enter a cavity, in which its extremity was free to move about, and pus immediately flowed therefrom.

A cannula having an aperture a quarter of an inch in diameter was introduced into the cavity by the side of the hollow needle, both being retained *in situ*, and through the former there took place a free escape of pus, along with some inspissated particles.

After the relief of the pressure the pus continued to well away more slowly, the respiration having a distinct effect upon the flow. At each inspiration it would become very slow or cease to escape; at each expiration the flow would suddenly increase. The pus was of a dark-brownish colour, evidently from the presence of disintegrated blood. The inspissated portions consisted of dark-green pus.

Two ounces six drachms of pus were collected, and fully two drachms of pus were lost, so that the abscess contained three ounces of pus.

The cavity was then washed out with one to forty carbolized solution, and to prevent undue pressure upon the delicate cerebral structure the stream was passed by cannula of small calibre into the cavity in the brain, from whence it issued by cannula of large calibre.

After all the particles had escaped, and when the solution returned tolerably clear and free from offensive odour, a stream of one per cent. carbolized watery solution was freely used. Then two decalcified chicken-bone drainage tubes were inserted, one projecting slightly into the abscess cavity, the other

extending an inch inside of it, and both were stitched to the external soft parts. A few stitches were introduced into the external wound, leaving the tubes flush with the scalp. Iodoform and naphthaline were dusted over the wound and an aseptic wood-wool pad was applied.

Immediate Effects of Relief of Pressure.—After the relief of pressure by the escape of pus from the abscess the ptosis of left eyelid became less marked; the left pupil, which had been hitherto in a state of stabile mydriasis, became mobile, and contracted to about medium size. The lividity of aspect and peculiar earthy hue disappeared quickly even while under chloroform.

Subsequent Course.—16th May, 1888 (twenty-four hours after operation). Patient slept most of the night. Several times when awake he spoke of his own accord, and seemed much more sensible and able to take an active interest in his condition. He asked for drinks, and the pain in the head had entirely disappeared. He became aware for the first time that he was in a place unknown to him—the Infirmary. He asked how and when he had come there, and seemed as if he had awakened from a dream. It was with difficulty he could be got to understand that he had been the subject of a severe operation. There was evident improvement of the oculo-motor paralysis, as indicated by the activity of the pupil and the slighter degree of ptosis. The grasp of the right hand had also improved by five kilos, as measured by the dynamometer; he was able to send it up to twenty (it was fifteen the day previous to operation). There was a marked absence of the fœtor which previously had prevailed round his bed.

17th May (forty-eight hours after operation). The muscles of the left eyeball had regained a considerable amount of their power, the eyeball being able to follow the finger upwards, downwards, and inwards, though not quite to the full extent. The ptosis had disappeared, the movement of the pupil was perfect. The power of pressure possessed by previously paralysed arm as indicated by the dynamometer was thirty, ten kilos higher than previous day and fifteen higher than before operation, but this power was very soon exhausted, a second trial giving twenty-five, a third twenty. The facial paralysis had considerably improved, the tongue was clean. He had had a good night's sleep, awoke refreshed, and asked about his friends. He expressed himself as quite well this morning, and ready for food. He wanted to rise.

18th May (seventy-two hours after operation). Patient slept all night and awoke refreshed, and was able for the first time to pass urine voluntarily. He drank milk freely. His pulse was seventy-eight per minute and his temperature normal. He could press the dynamometer to forty with the previously paralysed hand and to sixty with the sound one. He stated he was hungry, not regarding milk as food, and wanted to rise from bed.

21st May (six days after). He had perfect control over the muscles of the left eyeball. By means of a purgative his bowels moved freely for the first time since operation.

23rd May. *Ocular Examination*, by Dr. Reid. Left Eye.—"There are two small hæmorrhagic spots at the outer margin of the left disc. The

veins if anything are more congested, opaque, and tortuous than before operation. The disc, which was rather pale before, is now rather pinkish, with streaks of exudation at the upper margin of the disc, the refraction of centre of disc being equal to −4 D.'s. Right Eye.—The centre of optic nerve in right eye presents −3 D.'s of refraction; the veins, as in the other eye, are still congested and tortuous, the upper nasal being partly obscured by exudation as it crosses the margin of the disc. The arteries are small and shining in both eyes."

The patient henceforward pursued an uninterrupted course toward recovery, the only difficulty experienced being in restraining him from using too much liberty. His temperature never rose above 100° F., except on the night of operation.

At the termination of three weeks the dressings over the trephine opening were for the first time removed, when the wound was found healed. The decalcified chicken-bone drainage tubes had been absorbed at the skin line, leaving the minute portion which projected beyond the lips of the wound adhering to the wood-wool pad. The mastoid cells and antrum were occluded by a layer of granulation tissue, which was soon covered by epithelium.

Two months after the operation he was in excellent health, and his great increase in bulk formed a striking contrast to his former emaciation.

Remarks.—In inflammatory lesions of the temporo-sphenoidal lobe there are seldom any direct localizing symptoms. In the cases which have come under the personal observation of the author taste and smell have been regularly tested, and no satisfactory evidence of perversion of these senses have been detected, except with regard to taste when there was paralysis of the facial—chorda tympani. It is peculiar in this case that though patient on admission was in a dreamy, drowsy condition, he was yet able to answer questions, as was afterwards ascertained, correctly, as to his present state and past history, evincing both intelligence and memory, and he *appeared* as if conscious of his surroundings and *seemed* to comprehend the gravity of his position. Yet when he awoke from sleep the morning after the operation he realized for the first time that he was in a place unknown to him and had no idea of how he came to be in it. He was not aware that he had been removed from his own home. The last thing he did remember was that of lying in his own bed at home suffering from intense pain in the head. He had no idea of having given his consent to, or of having undergone an operation, and it was with difficulty he could be persuaded that he had had an operation performed on him. He believed that he had suffered from a sore head and nothing more. His psychical state, therefore, for some time prior to the operation resembled that of a somnambulist. A "dream state" has been noticed as occurring in cases in which the temporo-sphenoidal lobe has been the seat of tumours, though this "dream state" has not been specified to be of the somnambulistic order.

The temperature chart is appended.

CASE XXVI. *Case of cerebro-spinal lepto-meningitis, subsequent encephalitis and cerebral abscess in right temporo-sphenoidal lobe, arising from chronic otitis media. Repeated operations. Recovery. Patient admitted on the eighth day of his illness. Duration of illness seventy-seven days.*

P. M., aged 16, miner, admitted to Royal Infirmary, 25th September, 1891, suffering from cerebro-spinal lepto-meningitis, and subsequent encephalitis and cerebral abscess.

History.—*Heredity.*—He was one of a family of twelve, six of whom died in infancy. *Personal.*—He had measles and whooping cough, but otherwise had been healthy. Five years ago he had double otorrhœa, arising after bathing in dirty warm water near a coal pit. The otorrhœa has continued with short intervals since.

Present Illness.—Eight days previous to admission he was seized with pain in the right ear, which subsequently, and during the first twenty-four hours, extended into the same side of the head. He described the pain as excruciating, and during the time it continued he got no sleep. Shortly after the onset of the pain he had a severe rigor, which was followed by a persistent sensation of cold. During the first three days of his present illness he vomited repeatedly, his bowels were constipated, and he experienced a bad smell and taste, the latter of which caused him to spit constantly. On the fifth day of his illness his head became retracted, and the muscles of his neck were contracted, stiff and sore.

Condition on Admission.—He lay with his head and neck markedly retracted. The posterior cervical muscles were rigid, standing out like tense ropes. They were painful to the touch, the pain increasing on any attempt to flex his head. When he sat up in bed he had a tendency to fall backwards, and the rigidity of the head and neck became more apparent. He lay with his eyes shut, as the light annoyed him. His pulse was slow, from 56 to 60 per min. His temperature, however, was 101 ; pressure with the dynamometer was about 20 kilos and equal. His intellect was clear, and he answered questions intelligently.

Ears.—Hearing was defective in both ears. There was double infective otitis media, perforation of each membrana tympani, and foul-smelling pus issuing therefrom.

Eyes.—Optic neuritis was marked in the right eye, while the left was nearly normal.

Sensation was not affected, and there was no paralysis. The reflexes were almost normal, though occasionally there was slight ankle clonus.

Diagnosis.—Though the slowness of the pulse pointed to encephalitis, the other symptoms were those of cerebro-spinal meningitis, and with the object of relieving the latter the following operation was performed.

Operation (25th Sept., 1891).—The mastoid antrum was first opened. While doing this, the floor of the middle fossa of the skull was found to project more outwards and downwards than usual. So much was this the case, that in order to reach the antrum an aperture had to be made at a lower level than usual, and from that lower level the bone had to be

drilled in a forward and upward direction so as to curve round the base of the skull. After exposing the antrum it was found to be very much enlarged, and filled with granulation tissue and pus. The tegmen tympani was perforated, the perforation being recognized by means of a probe introduced from the antrum. The base of the skull was opened so as to expose the dura mater over the lower part of the temporo-sphenoidal lobe, fully an inch in length from before backwards, and for about the same distance from without inwards. The eroded and perforated tegmen tympani was removed. The whole of the dura exposed was thickened and inflamed, and pus which had been formed between the dura mater and the skull oozed out. All the pus, granulation tissue, and debris were carefully washed out, and an incision made through the thickened dura and pia mater, which gave vent to a small quantity of flaky serous fluid. The inner membranes were likewise inflamed.

Subsequent Progress.—After the operation patient's general condition greatly improved, the temperature fell, the rigidity of the head and neck disappeared along with the photophobia, but while this was the case his temperature and pulse soon became so low as to indicate encephalitis, his temperature being sometimes subnormal and pulse 48 to 60. He became extremely drowsy, sleeping a great deal. When roused he answered questions intelligently. The optic neuritis in the right eye instead of decreasing increased, while the pupil of that eye was somewhat dilated and sluggish. The left fundus was now somewhat congested. His expectoration increased, was slightly rusty, and had a foul smell. He had also slight increase of pain on the right side of the head above the ear.

These symptoms were taken as indicating encephalitis, and possibly the beginning of cerebral abscess. As he had basal lepto-meningitis and a pneumonic expectoration, it was thought right, as a preliminary to further procedure, to expose the dura over the sigmoid sinus and cerebellar fossa in order to ascertain definitely whether that fossa was involved.

Operation (3rd October, 1891).—This was done, when it was found that the dura over the cerebellar fossa was free from inflammation. A hollow needle was then introduced into the temporo-sphenoidal lobe from the aperture already made in the base of the skull, but there was no pus found. The needle, however, had removed brain substance which was in a state of acute red softening.

During the next four days patient's general condition at first slightly improved, but subsequently symptoms of the encephalitis increased. He had no shivering but was extremely drowsy, had a dreamy expression with slow cerebration, and his muscular power markedly deteriorated. It was probable that the area of cerebral softening had broken down into pus.

Operation (7th October, 1891).—The basal dura mater was again opened, and the cerebral tissue penetrated, when a copious discharge of pus mingled with shreds of necrosed brain escaped, probably to the extent of about three ounces. The cavity of the abscess was then thoroughly washed out by means of two cannulæ, the antiseptic fluid being introduced through

a narrow one and its exit taking place through another twice the calibre of the former. After the fluid returned perfectly clear the wound was dressed with iodoform and an aseptic pad, a decalcified drainage tube having been introduced through the outer wall of the abscess cavity.

Immediate Effect of Operation.—His pulse, which before the operation was extremely low, had improved on the table while the pus was flowing, and his temperature when taken after the operation had increased, as may be seen from the chart.

Subsequent Course.—After the exit of the pus his condition steadily improved. Within a few days his pulse and temperature were normal, his intelligence bright, and the whole of his former abnormal phenomena disappeared. His optic neuritis gradually decreased. On 1st November the right optic nerve was pink throughout, the cup nearly absent, vessels congested, with traces of exudation accompanying the inferior artery over the disc. In the left fundus the congestion was much less, as it had not suffered as the other had. His expectoration on 13th October was still profuse, and it had a distinct odour, but from that time it gradually lessened. He was so much improved that on this date (five weeks and two days after the operation) he was permitted to rise from bed.

Relapse and Formation of Second Abscess.—On the afternoon of the following day he complained of vertigo and headache, and next morning vomited. Subsequently he had pain in the right side of the head, after which he began to feel drowsy, was with difficulty roused, and had right external strabismus with right pupil medium and fixed. It was evident that there was further encephalitis, with probable involvement of the meninges, and therefore a hollow needle was on 15th November introduced through the aperture in the base of the skull into the temporal lobe, and withdrew therefrom half an ounce of pus. Owing to the previous aperture in the base of the skull being filled with fibrous tissue which had become firm and contracted, it was with difficulty penetrated and the cerebral puncture effected. It was therefore resolved, should the symptoms persist, instead of removing this tissue, to make a fresh aperture in the squamous portion of the temporal. Next day he was somewhat relieved, talked sensibly, was less drowsy, but there was no abatement of pain in the forehead and left ear.

Two days later the pain in the head and ears was greater, and his expectoration had increased. His right pupil was somewhat fixed and smaller than its fellow. It was evident that although patient had been somewhat relieved by the puncture and the withdrawal of the pus he was still suffering from encephalitis with abscess and a degree of lepto-meningitis. His temperature had fallen since last operation and his pulse become slower. He was exceedingly restless, constantly tossing about. The left side of the face first and the arm secondly became affected with paresis, which gradually increased, and amounted to absolute paralysis just prior to the operation about to be described. It was then questionable whether his left leg had become affected, but his mind at this time was so obscured that it was difficult to get him to perform any voluntary

movement, although he still continued to toss about with his right arm and leg.

Operation (19th Nov., 1891).—A disc of bone was removed from the posterior segment of the squamous just above the opening in the mastoid antrum. On the dura mater being incised a considerable quantity of pus spouted out, and when the brain was fully exposed the encephalitis was seen to be very extensive, and about three ounces of pus mingled with cerebral sloughs escaped. A large opening was made through the superficial brain tissue so as to freely expose the cavity of the abscess, when further sloughs were removed; the cavity was then well washed out, and a decalcified drainage tube was introduced.

Immediate Effect of Relief of Pressure.—The pulse rate while patient was under chloroform previous to the opening of the abscess, was somewhere about 60 per minute, but when the pus escaped it went with a bound to 114 per minute. The change was sudden, and exactly coincided with the relief of pressure.

Subsequent Course.—During the first part of the succeeding twenty-four hours he lay in a very exhausted state. He moved his right side freely, but his left remained powerless. For some days afterwards there was scarcely any improvement in the left arm though he moved the left leg quite as freely as the right. When he recovered the brachial power, the face still remained paralysed. All the ocular muscles supplied by the third nerve on the left side were paralysed. The pupil of the same side was dilated, evidently the third was implicated by basal meningitis on the left side. Neither pupil, however, reacted to light. The right pupil recovered first, and the recovery followed shortly after the relief of the pressure.

From this date he made an uninterrupted but slow recovery, though he had from the time of the last operation a small hernia cerebri owing to the red softening previously existing in the brain. With regard to the fundi they were examined about a fortnight after the last operation. The left was much as it had been at last note, but the right disc was more swollen than at any time previously, having a flame-like appearance. The veins were greatly distended, and the vessels were obscured by the exudation. On 3rd January, 1892, the condition of the eyes was as follows:—The right pupil was more dilated than the left, but both pupils reacted to light, and the muscles of both eyes acted naturally. The right fundus had its optic disc of greyish pink colour, the outline was everywhere distinct, but at its upper margin the exudation extended towards the retina, and hid the superior artery as it passed over the retina. The other vessels were of natural size, the superior artery was hidden all along its course over disc. The retina was natural. The left disc was greyish in colour, outline fairly distinct, except at superior and inferior poles where the blurring still existed. The vessels were not altered in size, the cup was filled in. The retina appeared natural.

Remarks.—This was a case in which the infective process had been grafted upon the old purulent otorrhœa, and had invaded the middle cerebral fossæ through the eroded tegmen tympani, inducing basal cerebral

CHART G.—SUPPURATIVE LEPTO-MENINGITIS AND RECURRENT CEREBRAL ABSCESS.

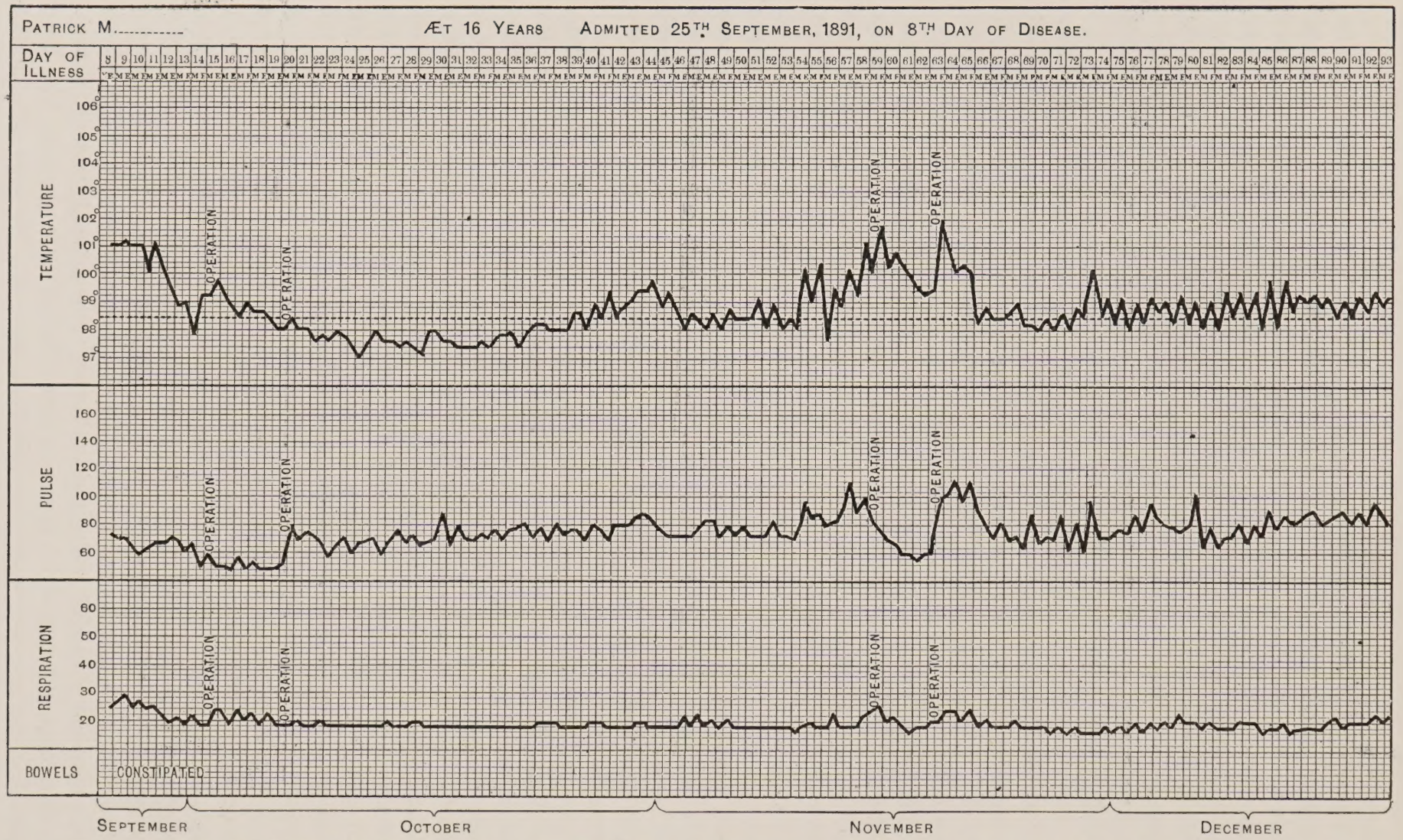


Chart of infective involvement of temporo-sphenoidal lobe. Suppurative lepto-meningitis followed by abscess, a second abscess forming during convalescence from the first abscess. The observations were recorded every four hours, but this chart represents the daily averages.

meningitis, probably implicating the upper part of the spinal cord and at the same time extending inwards, causing encephalitis and cerebral abscess.

At first the dominant symptoms were those of cerebro-spinal meningitis, judging from the marked retraction of the head and neck, with the contraction and rigidity of the posterior cervical muscles, the photophobia, the high temperature, and other initial symptoms. There was a substratum of encephalitis, indicated by the slow pulse, which, though it could have been produced by the meningitis, was much more likely the result of inflammation of the brain. The diagnosis of lepto-meningitis was supported by the actual appearance of the pia mater during the first operation, and the escape of the turbid serous fluid from the subarachnoid space, and, further, by the relief afforded by the removal of this secretion and the pus which was found between the skull and the dura mater. The rapidity with which all the symptoms of lepto-meningitis disappeared after the first operation may be regarded as affording hope for future cases of cerebro-spinal meningitis when operated on in their initial stage, especially when as yet the meningitis is confined principally to the base of the brain, pons, and upper part of the cord.

After the disappearance of the meningeal symptoms the patient still had a slow pulse, and gradually the symptoms of the encephalitis became slowly developed. The puncture of the temporal lobe at this period showed only red softening on the eve of pus formation, and within four days thereafter three ounces of pus along with sloughs of cerebral tissue were removed.

The cause of the formation of the second cerebral abscess five weeks after the first, while in the interval all the symptoms of encephalitis had disappeared, may be accounted for in several ways. Various causes may have contributed. He had in the opposite ear a purulent, probably infective discharge, from which pathogenic organisms may have gained access to the blood, and become deposited in the previously damaged brain tissue, where they found pabulum and germinating ground. It is also probable that several necrotic portions of brain tissue had been left, the micro-organisms being retained in the meshes of the slough as in a sponge, so that the brain tissue in the periphery of the former abscess, within the zone of softening, whose vitality had become impaired, had afterwards disintegrated or formed a slough round which the fresh abscess formed. The necrotic portions removed during the last operation were indicative of this.

The brain sloughs removed during this last operation were much larger and more numerous than any found in previous brain abscesses, and in this respect this case occupies a medium position between the cases of ordinary cerebral abscess and that of the necrosis of the temporo-sphenoidal lobe (Case XX.). The marked difference of the effect produced on the temperature and pulse by the lepto-meningitis on the one hand and the encephalitis on the other, is demonstrated by the accompanying charts.

The typical invasion of the motor area from pressure in the temporo-sphenoidal lobe was marked before the second operation, the paresis commencing in the face and passing into complete paralysis, as the initial

paresis showed in the arm. It was doubtful whether the leg of same side was affected ; if so, it was only to a slight extent. After the operation the paralyses passed off in the reverse order of their accession. In this instance both third nerves were implicated, the left by the extension of the basal meningitis. At the outset the right pupil was dilated and sluggish, and it was in this eye that the optic neuritis first developed. The occurrence of the after effects which the pressure of the abscess in the temporo-sphenoidal lobe might have had on the right oculo-motor was prevented by the basal meningitis.

It was observed that the optic neuritis between the first and the second operation increased instead of diminishing. It did so also at a very rapid rate, indicating an extension of the inflammatory mischief. Thus the optic neuritis gave a warning that the inflammatory action within the brain had not subsided. Occasionally one finds after acute abscess of the brain that the optic neuritis increases, but not to the same extent and degree as was evidenced in this case. The optic neuritis from its onset till its total disappearance lasted in all six months. This patient afterwards returned to hospital, when his other mastoid antrum was opened and the purulent otorrhœa cured. He was in perfect health at end of August, 1892.

CASE XXVII. *Cerebral abscess in temporo-sphenoidal lobe ; complicated with localized purulent meningitis of cerebellar fossa, both due primarily to chronic otitis media purulenta. Coming under observation seven days from onset of symptoms. Duration of illness sixteen days prior to evacuation of abscess. Operation. Complete recovery.*

H. M., aged 9 years, admitted into the Children's Hospital, Glasgow, on 26th January, 1893. Affected with purulent meningitis, accompanied by encephalitis terminating in cerebral abscess, involving the greater portion of the right temporo-sphenoidal lobe, due primarily to otitis media purulenta, an injury being probably the determining cause.

History.—*Heredity.*—Both parents are alive and well. Patient is one of a family of four, one of whom died when three months old from "inflammation of the brain." Other two are healthy.

Personal.—The parents believed patient to have been in good health previous to this illness. Though they had never observed discharge from his ears, yet on admission his right external ear was found to contain fœtid inspissated pus.

Present Illness.—Fourteen days before admission the patient fell head first down a flight of stone stairs. He rose immediately, apparently uninjured, and went about as usual. A week afterwards he complained of a severe pain in his head in and around the right ear, which compelled him to remain in bed. Since then he has had complete anorexia. Three days previous to admission, the pain extended to the frontal region. On the following day he vomited repeatedly, and a swelling was observed over the right mastoid. He has had constipation of bowels for several days, and has urinated involuntarily.

Condition on Admission.—Patient was in an irritable state, semi-insensible and delirious, but could be occasionally roused to realize his surroundings. He frequently cried out and tossed about, and was evidently suffering pain, referred to his right ear and corresponding side of the head. His pupils were equal, contracted, and sluggish. The movements of the eyeballs were free, and there was occasionally a slight transient convergent squint of right eyeball. There was a degree of congestion of both retinæ. A fluctuating swelling was observed behind the right ear over the mastoid. The right external auditory meatus contained foul smelling pus, and the membrana tympani was perforated. With the exception of the paresis of the right external rectus, there was no observable paralysis. There were very slight erratic twitchings of groups of muscles of limbs, mostly on the left, but also on the right side of body, and equally on both sides of the face. There was an herpetic labial eruption. His temperature was high, and his pulse rapid. On admission an incision was made to relieve the sub-periosteal mastoid abscess, and vent was given to about three drachms of fœtid pus.

Operation for Meningitis (28th January).—Two days after admission, the temperature having remained high, the pulse rapid, and the meningeal symptoms persisting, along with an increasing and now marked retraction of the head, delirium and other indications of brain involvement, the following operation was performed.

The mastoid antrum was opened. It was found to be enlarged, filled with cholesteatomatous masses and pus. The cerebellar fossa was then opened. The sigmoid sinus was covered with pus and a mass of granulation tissue, varying from $\frac{1}{8}$ inch to $\frac{1}{4}$ inch in thickness. Though its lumen was markedly encroached on, it was still patent. Granulation tissue also covered the dura behind the sinus in the cerebellar fossa, and several drachms of pus were evacuated from between the skull and that membrane. The pus from the cerebellar fossa was submitted to bacterial examination, and both by direct stained specimen and culture experiments the streptococci pyogenes were obtained. The tegmen tympani was carefully scrutinized, but no erosion was discovered.

Relief to all the meningeal symptoms followed this operation. The temperature, pulse, and respiration all became normal. The muscular tremors, the crying fits, and the delirium ceased, the pupils resumed their normal mobility, and on the third day the last evidence of the retraction of the head and neck disappeared. Following this improvement, it was observed that the pulse and temperature became subnormal. On the fifth day after the operation he became drowsy, disinclined to speak, and when both eyes were exposed to a light, the right pupil was a shade larger than its neighbour and distinctly more sluggish. All these symptoms increased until the ninth day after the operation when the evidence of pressure on the brain was marked. He was then extremely drowsy and difficult to rouse. There was a passivity over the left side of the face, and the left arm when lifted up dropped in a heap. The right arm, though feeble, the patient still controlled. Both lower limbs moved actively when the toes were pinched. An operation for the

relief of cerebral abscess was resolved upon. The symptoms pointed to abscess in the temporo-sphenoidal lobe; but as there had existed so much purulent meningitis in the cerebellar fossa which the former operation had revealed, it was deemed expedient to first subject the exposed cerebellar area to a careful scrutiny.

Operation for Cerebral Abscess (6th February, 1893).—The already exposed sinus was first inspected, and as some pus still exuded from the granulation tissue covering the dura behind the sinus, a quarter inch disc of bone was removed from the occiput covering the cerebellar fossa. The anterior half of the aperture left by this disc was covered with granulation tissue, while the posterior half exposed normal and almost transparent membranes, through which the underlying brain was just perceptible. There was no haziness or milkiness of the membranes over this area, such as would have been expected had there been an extension of the meningitis in this direction. Attention was now directed to the temporo-sphenoidal lobe, and as a preliminary a fresh scrutiny of the tegmen tympani and tegmen antri was made, but no visible erosion was detected.

The incision which had previously exposed the mastoid antrum was extended upwards over the squamous plate of the temporal, and from it a half inch disc of bone was removed from a point half an inch above and a little behind the external auditory meatus. At this point the dura was healthy, and so transparent that a flake of plastic effusion $\frac{1}{8}$ inch in diameter was seen either overlying or within one of the compressed pial vessels. This speck of effusion could be altered in position by probe pressure applied from the external surface of the dura mater. The surface of the brain was markedly avascular, probably due to the pial vessels being flattened against the dura and skull from intracranial pressure, but possibly from thrombosis of some of the larger trunks. When the membranes were incised the avascular brain bulged into the trephine aperture. A hollow needle was introduced into the temporo-sphenoidal lobe in an inward and downward direction; when it had penetrated the brain substance for about half-an-inch, there was first the escape of some gas followed quickly by a jet of pus, greenish in colour and of foul odour. A pair of forceps were afterwards introduced through the healthy brain tissue and expanded, when the pus was given free vent, and with it there came a series of cerebral sloughs, some of considerable size. The amount of pus and disintegrated cerebral tissue, though not accurately measured, was approximately about three ounces. After this matter was removed and the brain tissue held aside, a large cavity was seen to exist. Its limit in the anterior and posterior directions was not visible from the trephine opening, its depth was fully an inch, and a probe could be passed backward for about two inches and forward for about one and a half inches before coming in contact with brain matter. Considering the size of the child's head and that of the cerebral cavity, the greater portion of the temporo-sphenoidal lobe must have been obliterated. The brain tissue exhibited very little tendency to fill the gap, which remained as a cavity of the above dimensions at the end of the operation. The cavity was not syringed, lest infective particles might be disseminated especially

into the lateral ventricles ; but it was evacuated as thoroughly as possible, the head being placed in several directions to facilitate complete evacuation by gravitation. Iodoform and boracic powder were then introduced into the cavity, a short decalcified drainage tube was fixed in it by means of a stitch, and a wood-wool dressing was applied.

The pulse, which ranged from 56 to 70 before operation and was 72 before the evacuation of pus, increased in rapidity immediately after the operation, and when the patient was removed to bed was to be found to be 104, and two hours afterwards 116 ; subsequently it subsided to normal. The charts show the temperature, pulse, and respiration during the whole course of the illness.

Course after Operation.—From this date the patient made an uninterrupted recovery. He was dressed for the first time two weeks after the operation, and again at the end of a month from the date of the operation. At the former dressing the brain cavity no longer existed, and only a minute portion of the drainage tube lay exposed on the granulating surface, the remainder having been absorbed. The external wound healed by granulation tissue.

CASE XXVIII. *Abscess in temporo-sphenoidal lobe due to infective otitis media. Doubtful condition of lateral sinus : lungs involved. Coming under observation on fifth week of illness. Operation. Complete recovery. Duration of disease six weeks.*

W. H., a boy aged 9 years, was admitted into the Hospital for Diseases of the Ear, under the care of Dr. Barr, on 13th January, 1887, suffering from infective purulent otitis media of right ear and abscess of the brain. For the diagnosis and treatment of the latter the writer was ultimately consulted.

History.—*Heredity.*—Family healthy. *Personal.*—The history previous to his admission into the hospital, as given by his mother in answer to questions, was as follows :—

A month prior to the operation about to be described, he complained one day of pain in the region of the right ear, which made him fret, and prevented him from sleeping at night. On the following morning his skin was hot and dry, and he vomited, after which he became drowsy, being frequently awakened, however, by sudden startings and crying fits, due to acute pain. The pain over the region of the ear, the drowsiness, the feverishness, and the occasional vomiting continued to characterize the first seven days of his illness. On the eighth day, he had a rigor which lasted a quarter of an hour, in which he shook all over and his teeth chattered. Between this time and his admission into the institution he had in all six rigors, at intervals of about two days. On the nineteenth day of his disease he was admitted into the Hospital for Diseases of the Ear.

His condition was then as follows :—He was a greatly emaciated child, having a pale, shrunken face with livid lips, his upper one being covered by

a herpetic eruption. He had a short, catching cough. His axillary temperature was 100·8° F. Pulse 108. He was very drowsy, complained of pain over the right ear, from which there was an offensive discharge.

Dr. Barr found a perforation to exist in the upper part of the tympanic membrane, from which some purulent secretion was escaping, although the quantity was not great. There was defective hearing on that side, but the boy's mental condition prevented an exact estimation of the degree of deafness.

For two days after admission (twentieth and twenty-first days of his illness) his complaints of pain were not obtrusive, but the drowsiness continued. On the third day after the pain had become aggravated, he had a slight rigor. There was no evidence of an acute inflammatory process, or of retention of pus in the ear, to account for the pain, and there was neither redness nor swelling over the mastoid process. Temperature at this time was 99·6° F. ; pulse 92.

Dr. Barr perforated the mastoid process, opened into the mastoid cells, and succeeded in passing a stream of antiseptic fluid through the antrum and tympanic cavity out by the external meatus. A small quantity of purulent and caseous debris was washed out. A drainage tube was introduced into the orifice in the bone, through which an antiseptic solution was injected twice daily, from the opening behind to the external meatus and *vice versa*. For two days subsequently no change was observed in his condition, but a slight rigor occurred on the second day after the operation (twenty-fifth day of the disease), while the pain in the head and the drowsiness became worse. On the twenty-sixth day of his illness, a copious discharge of most offensive odour escaped from the ear. His tongue was red and smooth, and he refused to take food or drink. Pulse was intermittent and irregular. A red flush frequently suffused the face.

On the thirtieth day of his illness Dr. Barr consulted the writer as to the presence of cerebral abscess. The condition of patient when seen was as follows :—On approaching his bed a heavy fœtid odour, traceable to the discharge emanating from his right ear, was perceived. He was an extremely emaciated child, with pale greyish face, wrinkled skin, prominent cheek bones, and somewhat sunken eyeballs, who lay upon his right side in bed, his hand beneath his head, looking as if he were asleep with his eyelids half open. His lips were livid, and on the upper one remains of a herpetic eruption appeared. His tongue was red and dry. Sordes covered the teeth.

When an attempt was made to rouse him the right upper eyelid drooped, and there was a shade of passivity over the left side of the face, with a slight dragging of the angle of the mouth toward the right. The conjunctiva of the right eye was congested, and the eyelids were smeared with pus. The pupils were equal, about medium size, and responded sluggishly to the action of light.

The veins over the right side of the head were congested, and stood prominently out on his shaven head. Pain on percussion was elicited over the right temporal, while percussion over the head generally was calmly borne. There was no œdema about the mastoid process. The right sterno-

mastoid muscle was rigid. There was no hard cord-like feeling along the line of the internal jugular, though the external jugular was prominent. Acute pain was elicited on deep digital pressure just behind the origin of the sterno-mastoid, in the tissues near the exit of the vein which passes through the posterior condylar foramen.

He was in a state of stupor from which he could only be partially aroused, and not to such an extent as to give an intelligent answer to any question. His pulse was markedly slow, 50 to 60 per minute, and was feeble and intermittent. His limbs were extremely attenuated, and there was an irregular muscular tremor over the body generally, such as one frequently finds in approaching dissolution. He had a frequent cough with purulent expectoration, which had an offensive odour, and there were moist râles over both sides of the chest. His bowels were and had been obstinately constipated. His urine was free from albumen. It was somewhat difficult to believe that a few weeks previously this boy had been apparently stout and robust, and had been vigorously engaged at football. There was a flow of excessively fœtid fluid from the external ear, and as this was greater in amount than what would be likely to come from the antrum or middle ear, it was suspected that there might be a connection between the tympanum and the intracranial cavity.

There was no difficulty in concluding that this patient suffered from cerebral abscess, originating in infective otitis media. It was almost certain that it was situated in the temporo-sphenoidal lobe, and it probably had a tortuous and minute communication with the middle ear. It was feared that the lung affection was secondary to the infective absorption.

The prognosis was almost hopeless, his youth being the one favourable point. Immediate operation was advised, and with Dr. Barr's concurrence it was at once carried out.

Operation.—The middle ear was washed out with an antiseptic solution, and rendered as nearly aseptic as possible. A half-inch disc of bone was removed from the squamous portion of the temporal, at a point an inch above and half an inch behind the centre of the external auditory meatus. The bone was normal. The dura mater was here slightly congested, and did not impart any brain impulse to the finger; neither was any discernible when the aperture in the skull was filled with fluid.

When this membrane was opened and turned aside the brain tissue immediately bulged into the osseous aperture, and rose above its external level. The pia mater covering it was congested, and the brain substance had a yellowish red appearance.

A hollow needle was inserted into the brain, in a direction which, if introduced far enough, would strike the eminence in the petrous bone, above the middle ear. After it had penetrated the brain tissue for about three quarters of an inch there was a sudden discharge of foul gas, accompanied by a bubbling sound and the escape of a few bells of air and fluid. Evidently the upper part of an abscess cavity had been tapped, and this contained fœtid gas. The needle was inserted a little further, when the pus flowed out. It was of ordinary yellowish colour, and had a most offensive odour.

After two drachms had slowly welled away, the aperture in the brain tissue was enlarged by forceps, and portions of the brain tissue which had become necrosed were removed. These last consisted of shreds, two about the size of large horse beans, the others that of barley grains. On their removal more pus welled away. The cavity was then washed out with a saturated solution of boracic acid. After that, however, there was still a slow oozing of pus, along with extrusion of minute shreds of brain tissue.

It was evident that there was a considerable zone of purulent inflammation surrounding the abscess cavity, from which the pus continued to ooze. An aperture was then drilled into the base of the skull, through the tegmen antri and tympani, involving the petro-squamosal suture. The dura mater was examined here and found to be thick and pulpy. It was penetrated, and the abscess cavity was reached. A stream of boracic lotion was passed from this aperture, so as to wash out the cavity of the abscess, and it was continued until it passed freely out by the upper opening. The current was then reversed. Even after that, when the drainage tube was inserted, an oozing of purulent fluid took place into the tube.

The ear was again cleansed. Chromicized chicken bone drainage tubes were introduced into both apertures. The parts were thickly dusted over with boracic acid, and dressed with sublimated wood-wool pads. The disc of bone was not reimplanted.

Subsequent Progress.—When placed in bed patient was extremely weak, but rallied after the first few hours. A marked improvement was observable during the first week. The pulse became quicker, the temperature a little higher—both good signs; the face became brighter, the eyes clearer, the conjunctiva free from pus, the ptosis vanished, and the mental condition improved. After the first week, though kept on restricted diet, he improved in weight, and continued subsequently to do so at a rapid rate.

Dr. Reid examined the eyes about the end of the fourth week after the operation, and could find no trace of optic neuritis, and was further of opinion that optic neuritis could not have been present at the time of the operation without having left some traces of its presence four weeks after.

It is probable that the aperture in the tegmen tympani and membranes led into the abscess cavity, from which the larger amount of pus noted escaped before operation.

January, 1892.—The boy was still strong and well, suffering no inconvenience from the pulsating hiatus in the skull. This soft part ultimately became hard from contraction of the connective tissue and bone thrown out from the sides of osseous aperture.

CASE XXIX. *Cerebral abscess in temporo-sphenoidal lobe due to infective otitis media. Operation (in Dr. Macewen's absence) by Dr. Clark. Recovery. Coming under observation on the thirtieth day of his illness. Duration of illness one hundred and twenty days.*

T. G., aged 16, lithographer, admitted to the Royal Infirmary, Ward 21, 19th April, 1891, with abscess in the left temporo-sphenoidal lobe.

History.—*Heredity.*—Family history was good. He was one of twelve, nine of whom are alive and well. Two died from measles in infancy. There was no tubercular history. *Personal.*—Had been in good health, with the exception of the condition of the ear and head.

Present Illness.—A month previous to admission, he had been playing at football when he was believed to have caught cold. This was followed by pain in the left ear and left side of head which lasted four days, and then extended to the front of the head, and became of a throbbing character.

Six days from the commencement of the illness, he began to "wander mentally," and had a difficulty in memory. On that day also it was noticed that he had a purulent discharge from the left ear, and a swelling appeared in the temporal and mastoid regions, extending down to the angle of the jaw. An incision had been made into this, after which the swelling subsided, the patient experienced great relief, and was able to rise from bed and take food with tolerable comfort. He continued fairly well, till thirty-six hours before admission (twenty-eighth day of illness), when he was unable to rise from bed, was sick and vomited, and gradually passed into an unconscious condition. During the last few days, although his friends state that they did not observe shiverings, still he had profuse sweats, which probably followed rigors. There was no defect of eyesight as far as could be observed, and he did not appear to be deaf. His bowels had been rather constipated.

Condition on Admission.—Patient was in an unconscious state, it being impossible to rouse him, though he winced on firm pressure over the mastoid process.

The body was fairly nourished ; surface pale and moist. The limbs of the left side were capable of voluntary movement, but those of the right lay flaccid, and were not moved. The pupils were unequal, the left was dilated and fixed, the right was medium and responded feebly to light. Respiration slow and sighing (12 to 15 per minute). Pulse slow and markedly dicrotous (65 per minute). Temperature 98·8° F. Ankle clonus easily produced. Knee jerks not developed (probably through faulty manipulation), superficial reflexes active.

Operation (20th April, 1891).—As the case was urgent Dr. Clark operated in the absence of Dr. Macewen. The mastoid cells were first cleared out. Communication with the middle ear was not established, and no pus was obtained from the mastoid. He next trephined over the temporo-sphenoidal lobe, and on removing the disc of bone, the dura mater, soft and discoloured, was observed to bulge through the opening. The dura mater being slit, a small cannula was introduced, when half an ounce of greenish foul-smelling pus escaped. The abscess cavity was well irrigated and a bone tube introduced. The patient's condition improved greatly after the evacuation of the pus.

Subsequent Course.—For the first two days after the operation, the patient remained in an unconscious condition, passing urine in bed, moving the left arm a little while the right remained flaccid and motionless, eyeballs turned to the left, both legs becoming occasionally rigid for about

ten minutes at a time. He swallowed with difficulty, probably owing to his unconsciousness. His skin was covered with cold perspiration, and there was slight purulent discharge from both eyes. Long sighing respirations occurrred occasionally.

About a week after the operation, he began to have glimmerings of intelligence, he swallowed more freely, and on several occasions he moved the right arm. He still passsd urine in bed. Ten days later, the left pupil which had throughout remained dilated and somewhat fixed became much more widely dilated than before. He was very restless, and constantly tossed about as if experiencing pain, although his intelligence was not clear enough to indicate its locality. His temperature at the same time dropped to about 98°, 99° F. His pulse also came down to about 54.

On the twenty-fifth day from the operation the restlessness became less, his pulse improved, and his intelligence became markedly better. The right arm occasionally moved voluntarily. On the twenty-eighth day he was able to tell his name and answer simple questions, but still he had restless attacks lasting several hours. During all this period there was free suppuration from the wound in the brain, necessitating dressing every alternate day. About the twenty-eighth day after the operation the right arm was affected with constant jerking movements, somewhat choreic in character, and these persisted till 5th July, seventy-six days after admission. About sixty days after operation he had improved much in general condition, taking his food well. His emaciation rapidly disappeared, and he got so much stronger that he was able to rise from bed, and his intelligence becoming much more active he was able to express himself.

During this period the reflexes were found to be exaggerated, ankle clonus was marked, and the knee jerks much increased. On rising he walked with a peculiar gait, both legs being affected, but the right markedly so. The affection somewhat resembled a paraplegia, but with a degree of choreic-like movement.

Relapse and Formation of Second Abscess.—At 11 A.M. on 5th July, the seventy-sixth day, he took a series of convulsions, ushered in by the head being turned to the left, the mouth and eyes being turned to the same side ; his limbs were affected in the first convulsion without any precise order. In the subsequent fits, which occurred at short intervals, the right side of the face, the eyes and mouth twitched to the right, the right arm and right leg following, and the rest of the body then becoming affected. He lost consciousness during these fits. In the last attacks this order was lost, the convulsions appearing irregularly.

Operation.—The wound, which had been kept open, though covered with granulation tissue, was penetrated with a pair of dressing forceps, and about two ounces of pus escaped.

The temperature, pulse, and respiration chart showed that these were all above normal, and continued uninterruptedly so for about eight days, when there was a temporary fall, which became marked two days subsequently, the pulse and respiration becoming sub-normal, and continuing so with slight variations for some weeks.

Five days before the convulsive attack his temperature was slightly elevated, his pulse rate greatly increased, and the respiration hurried.

An hour before relieving the abscess on the 5th July his temperature was 104°, after about five hours it fell to normal. Subsequently, with the exception of the rapidity of pulse, there was no abnormality recorded.

Remarks.—It was evident that this patient was affected with meningitis as well as with cerebral abscess. The rigidity of both legs spoken of, the squint and purulent discharge from the eyes, the constant restlessness and tossing about, the irregular choreic-like movements of the hand and leg, the high temperature, and rate of pulse, and respiration, all pointed in that direction. The meningitis had extended probably some way down the spine. Patient subsequently recovered perfectly, and was dismissed well.

Since patient left he had been in good health, and was regularly at work until the 28th May, 1892 (thirteen months after operation), when he returned complaining of having had a "fit" after being over-heated at football. No medical man saw the fit, therefore it was not described, but he was kept in the ward for a week and showed no further symptoms. The power of his arms was tested by the dynamometer, the left being a little stronger than the right, the pressure being about 30 to 35 kilos.

CASE XXX. *Cerebral abscess due to chronic infective otitis media, with caries of left petrous bone. Abscess situated in the left temporo-sphenoidal lobe, the base of motor region becoming implicated. Marked and sudden change in symptoms indicating bursting of abscess into ventricles. Trephining during deep coma, while patient in extremis, evacuation of much pus from brain, and removal of encapsulated abscess. Whole temporo-sphenoidal lobe destroyed. Relief of symptoms. Death from exhaustion. Post-mortem and examination of cyst removed from brain during life. Duration of illness indefinite, last phase over fourteen days.*

C. S., æt. 14 years, was brought under observation on 31st October, 1881, suffering from symptoms of intracranial suppuration, arising from otitis media, and caries of the left temporal bone.

History.—*Heredity.*—Nothing was ascertained concerning her family. *Personal.*—The history of her illness could not be learned with any approach to accuracy, as there was no source from which reliable data could be obtained. The following points were however ascertained.

A fortnight prior to the above date, she had been at work, and while there, had complained of pain over the left side of the head, accompanied by a feeling of sickness and prostration. A week subsequently a surgeon opened an abscess, situated over the left side of the head, which pointed above and in front of the left ear, and from which a considerable quantity of pus was evacuated. No information could be obtained as to the date on which the purulent discharge from the ear commenced, but from the condition of the parts it was obviously of old standing.

Condition on Admission.—The girl lay on her left side in bed, her head resting on her left hand, which lay on the pillow. Her body was bent forward, and her knees were flexed on the abdomen. Her features were pinched, and *cutis anserina* was marked on her legs and arms. She tended to doze persistently, though she was restless and evidently disturbed. She frequently shrugged her shoulders, muttering the word "cold" and attempting, often abortively, to cover herself with more blankets, even when they were heaped over her. There was no actual rigor during the short time she was under observation. She had right facial and brachial paralysis, and paralysis of all the branches of the left oculo-motor. All her movements were slow and feeble. Her sensory condition could not be definitely ascertained. Her pupils were unequal, the left being dilated and fixed. There was slight conjunctivitis, and there was marked optic neuritis. She was absolutely deaf on the left side, as tested by both aërial and bone conduction. She heard well on the right side. On the left side the membrana tympani had disappeared, the cavity of the tympanum being occupied by granulation tissue and fœtid pus. The petrous portion of the temporal was affected with caries, and there was bare bone over the squamous and mastoid portions. Her cerebration was extremely sluggish. When interrogated, she turned the sound ear toward the speaker, smiled vacantly, and after an interval replied in a monosyllable, not always intelligibly. She would then drop off into a doze. The pulse was markedly slow, temperature subnormal. Respiration slow, urine highly albuminous. It was diagnosed that patient suffered from cerebral abscess, located in the left temporo-sphenoidal lobe. Shortly after this examination fresh phenomena occurred in the shape of a series of spasmodic contractions affecting the muscles of the right side of the face, and several hours subsequently extending to the right arm. These were on the opposite side of the body from the brain lesion. From these it was inferred that the inflammatory zone round the abscess had by extension invaded the lower portions of the motor area on the left side of the brain.

Though patient was in a very weak state, it was resolved to trephine through the squamous portion of the temporal, so as to expose the temporo-sphenoidal lobe. This operation was fixed at morning visit to take place at two p.m. on same day, 1st November.

At nine o'clock on that morning patient was in much the same state as has just been described. The respirations were subnormal, the pulse weak and markedly slow, its rate being not more than forty per minute, and the rectal temperature was also subnormal. About one o'clock on that date a marked change commenced, the various phases of which occupied about an hour in fully developing. Slight twitchings of the muscles occurred all over the body. These followed no definite order. They occurred almost continuously, and were irregularly distributed. Ten minutes after their onset, the patient became gradually unconscious, and was soon sunk in deep coma. The respirations became very rapid, laboured and occasionally sighing. The pulse was 140 per minute, and very feeble. The temperature, which had been taken every three hours, registered 100·6° F. at ten a.m.;

103·2° at twelve ; 105·8° at ten minutes to two p.m. At that hour the face was deeply livid, and the pupils were widely dilated and fixed. This sudden change was believed to be due to the bursting of the abscess into the lateral ventricle.

Although operation at this stage was a forlorn hope, it was considered right to give vent to the pus.

Operation.—Patient was so deeply comatose that no anæsthetic was required. A disc from the squamous portion of the temporal was raised an inch above the external auditory meatus. The dura mater was seen to be congested, opaque, and thick. There was no pus in the subdural space. The pia mater was œdematous, deeply congested, at parts thickened, and here and there deposited upon it were minute yellowish-white flakes of lymph. The surface of the brain had a deep red appearance, and rose to the upper level of the opening in the skull, slightly bulging beyond. There were absolutely no cerebral pulsations. On palpation with the tips of the two index fingers, fluctuation could be discerned, though not very distinctly. An incision was made into the brain substance for about half an inch, when a jet of pus, having a fœtid odour, spurted out over the hand of the operator. The incision was enlarged, when about three ounces of dark yellowish brown foul-smelling pus escaped. In looking into the cerebral abscess cavity, the inner wall of which was red, shreddy and flocculent, it was observed that a white cyst-like structure lay toward the bottom of the space. It was quite detached and floated about in a quantity of pus which remained. It was gently touched with a pair of forceps, when, finding it to be quite loose, it was lifted out. As this cyst floated about in the pus within the cranium, it had the form and appearance of a tennis ball. When all the pus had been removed from this aperture, the great gap revealed in the brain was of very serious import.

Before the abscess was opened, and while the operation was proceeding, patient's pulse had become imperceptible, the lividity of the face had increased, and there were ominous pauses between the respirations. Immediately on the abscess being opened there was a marked change in these respects. The pulse from being imperceptible speedily returned and was soon markedly reduced in frequency, registering about 100 per minute. The respirations greatly improved, the lividity of the face vanished, and in its stead something of the former pallor returned, though now there was more colour in the lips. Patient became sensible to pain, and consciousness was partly restored.

She improved in all these respects for about a couple of hours after the operation, and hopes were beginning to be entertained of the possibility of her recovery. Shortly after, however, the feebleness from which she suffered became greater, excessive prostration ensued, and she died six hours after the operation.

Post-Mortem Examination.—On the calvarium being removed, the dura mater over the left side of the cerebrum was much congested and thickened, while that portion of it which corresponded to the temporo-sphenoidal lobe was covered by a layer of lymph. When the membranes were reflected

the abscess cavity was seen to occupy the whole of the temporo-sphenoidal lobe, as well as a very small portion of the base of the parietal, and to have encroached on the left ventricle by a distinct breach in its wall. The walls of the left ventricle were much softened and flocculent. The right ventricle was distended, principally with serum, but also contained a little pus, possibly half a drachm. The cerebral tissue, for fully an inch round the abscess cavity, was of a reddish yellow colour, softened, pulpy, having undergone purulent infiltration. The wall of the abscess was flocculent and shreddy, some of the thrombosed vessels lying in the cavity like threads loose at one end. The right side of the brain seemed little affected and the membranes of that side surprisingly so when the amount of damage to the opposite side was considered. The left petrous bone was in a state of extensive erosion, especially in line with the petro-squamosal suture. The erosion communicated with the middle ear, a distinct circular aperture existing at this point measuring a quarter of an inch in diameter, and looking as if it had been drilled. Filling this osseous gap in the tegmen tympani was a mass of well-formed connective tissue attached to the dura, which projected externally into the middle ear, and internally through the membranes into the abscess cavity. Though a probe could not be passed through this connective tissue mass, yet when the nozzle of a syringe was inserted through a nipple-like projection on its inner surface a few drops of water could be forced from the abscess cavity into middle ear. Judging from the appearance of the connective tissue, it was evidently of very old standing. From the fact that its nipple-like projection protruded into the abscess cavity fully quarter of an inch from the level of the dura mater, and also from the fact of its matured formation, it was probable that the ball-like cyst—evidently a chronic abscess—had been attached to this connective tissue mass. It was also probable that this had been an outlet for the escape of pus from the brain, an '*otorrhœa cerebralis*' —only the abscess in the brain was secondary and due to disease of the middle ear.

The erosion of the petrous bone did not end here, it extended forward and perforated the temporal, into the glenoid fossa near the fissure of Glaser. A small quantity of pus was found in the joint of the lower jaw. Between the eroded bone and the thickened dura there was a layer of pus. The other internal organs were anæmic, but showed no trace of inflammation. The lungs were slightly congested.

Examination of the wall of the cyst-like abscess, removed from the brain at operation, and after prolonged steeping in spirit.

The cyst, which was of an oval shape, measured two and a half inches in length by one and a half in breadth (62 mm. by 37½ mm.). Its walls were of variable thickness, at one part about five millimetres, at another about two, though for the most part it measured about three. The dimensions were considerably reduced by prolonged steeping in spirit. Its outer surface was very flocculent, while its inner was lined with a smooth glistening polished layer, about a millimetre in thickness, which could be peeled off, leaving a rougher layer underneath. On microscopical examina-

tion of a portion of this abscess wall, there were masses of granulation cells (leucocytes), a few connective tissue striæ, a number of spindle cells occurring in groups, and interspersed throughout were masses of degenerated cells and granular debris. There were also a very few bloodvessels of primitive formation, such as are met with in granulation tissue. (See Figs. 45 and 46 in chapter on Pathology.)

Remarks.—This case was primarily one of otitis media, leading to caries of the temporal bone, with subsequent formation of cerebral abscess. The pus from this abscess had in all probability found its way through the membranes, and had been discharged by the carious aperture in the tegmen tympani, which communicated with the middle ear. This would occur by small quantities oozing through the granulation tissue. By a more vigorous growth of fresh connective tissue the exit aperture had become occluded, especially as the amount of pus produced by the abscess had diminished. Then the wall of the chronic cerebral abscess became complete at the parietal side, as it was at the visceral. This might account for the greater thickness of the abscess wall on the visceral than on the parietal aspect. A so-called "latent period" ensued, during which patient resumed her work. The form and appearance of the abscess cyst would indicate a probable duration of some years. The meagre scraps of history obtainable show that this so-called latent period must have lasted many months at least. Recently a fresh attack of infective inflammation occurred in the periphery of the encapsulated abscess which destroyed the whole temporo-sphenoidal lobe, and even encroached upon the parietal, and finally burst into the lateral ventricles. The cause of the fresh attack was obscure. It could have occurred through fresh infective invasion springing from the middle ear, or from the bursting of the encapsulated abscess wall, arising from a blow or injury to the head, the extravasated pus setting up fresh inflammation in the brain tissue, reduced in vitality by the presence of a foreign body, as the encapsulated abscess may be regarded.

CASE XXXI. *Chronic cerebral abscess in temporo-sphenoidal lobe due to infective otitis media. Abscess cavity communicated with middle ear. Acute inflammation in brain following injection of nitrate of silver into external ear. Death. Seen in extremis. Duration of abscess probably at least one year.*

In April, 1883, the writer was asked to see in private a case of cerebral abscess under the care of Dr. Scott, of Tollcross. The patient, **A. L.**, a boy of three years, had had a purulent discharge from his left ear for over two years. A year after the commencement of the discharge, he had had an "illness in his head" supposed to have been cerebral abscess, which lasted a month, and toward the end of that period a "great discharge" of pus escaped somewhat suddenly from the left ear, after which he rapidly recovered. From that time he continued in fair health, though the purulent discharge from the middle ear persisted. In the hope of curing this, the parents applied to a dispensary where a solution of nitrate of silver was introduced

into the ear. The mother stated that this solution when introduced did not return, but was retained, causing excruciating pain, followed in a couple of hours by delirium, and finally by coma, which persisted until death, which occurred four days after the injection. The child was only seen by Dr. Scott a day after the injection had been given, and by the writer a few minutes prior to death. After death an external examination showed that the membrana tympani had disappeared, the tegmen was ablated, an aperture existed in the tegmen tympani, the membranes were perforated, a probe passing freely into a cavity in the base of the temporo-sphenoidal lobe, from which pus oozed through the granulation tissue. This was no doubt an old cerebral abscess cavity with firm walls. Probably the nitrate of silver solution entered the cavity in the brain, causing intense encephalitis, and being the immediate cause of death. It is also probable that this abscess was of more than a year's duration, and that the large quantity of pus which came away toward the end of the month's illness, which occurred a year prior to death, came from the abscess cavity. No further examination was permitted.

CASE XXXII. *Note on a doubtful case diagnosed as cerebral abscess. No pus found at operation. Sixty hours later pus found in the mouth, the middle and the external ear, after which patient rapidly recovered.*

In 1889 a girl, **D. B.**, aged 13 years, a patient of Dr. Barr, was sent to the Royal Infirmary in an insensible state, suffering from cerebral symptoms, diagnosed by the writer to be probably those of cerebral abscess, and from the presence of left facio-brachial paralysis, and of right otitis media, it was considered to be located in the right temporo-sphenoidal lobe, which was consequently explored by the hollow needle, and no pus was found, though the membranes and brain surface were congested. The patient remained unrelieved for two days and a half. At the termination of that period, as she lay on her left side in an insensible state with the mouth half open, the space between the teeth and the flabby cheek was found filled with pus. Two tablespoonfuls of pus were removed from the mouth, at an interval of ten minutes between each. More pus may have been swallowed by the patient. There was at the same time an increased quantity of pus from the middle and external ears, the increase amounting probably to about two drachms. From this time patient rapidly recovered, the power of the left arm and face was gradually regained, and she was restored to complete health.

The case is still one of doubt, but from the escape of the pus and the coincident relief of the cerebral symptoms, it appeared that there had been a cerebral abscess, which probably had found an outlet through the tegmen into the right ear, and as she lay on her left side the pus may have gravitated through the Eustachian tube and so found its way into the mouth. If this were so, why did the hollow needle not relieve the abscess in the temporo-sphenoidal lobe? Experiments on brains with a hollow needle show that

the cerebral tissue penetrated by the needle occasionally fills its lumen, especially when the needle was given a slight rotatory turn during insertion. When this occurs the needle becomes blocked, and the pus is prevented from flowing through it. What was believed to have occurred in the above case actually took place in a very interesting case recorded by Mr. Field, in which he penetrated the brain in search of an abscess but found no pus. The patient died, and post-mortem examination showed that the instrument had not only penetrated but had actually transfixed the abscess cavity. (See means of avoiding this under chapter on Treatment.)

Localizing symptoms of abscess in the frontal lobes.—Abscess in the *frontal lobe* may be attended with no localizing phenomena, unless situated about the base of the third frontal, or the ascending frontal convolution, or implicating these by the zone of encephalitis which surrounds it. Pupillary manifestations are frequently present. If the abscess be large, the pupil on the same side is in a state of stabile mydriasis; if small it may be sluggish and contracted.

Transient aphasia with *post-convulsive right hemiplegia* diagnostic of abscess.

In an abscess sequent to infective traumatism, motor aphasia was a temporary but pronounced feature, accompanied by post-convulsive right hemiplegia. The abscess was situated in the white matter of the third and ascending frontal convolutions, and involved the conducting path of the fibres from the speech centre, causing motor aphasia.

Lesions in the conducting paths to the internal capsule from the cortical speech centre may produce only transient aphasia, the speech process being able to pass by strands to the corpus callosum and opposite speech region. See figure under Case XXXIV.

CASE XXXIII. *Cerebral abscess in left frontal lobe of traumatic origin. Symptoms of bursting into the lateral ventricles at termination of the eleventh week. Immediate operation, removal of three ounces of pus, relief of graver symptoms, persistence of slighter ones for a week thereafter. Complete recovery. Coming under observation ten weeks after injury. Duration of disease eighty-three days (about twelve weeks).*

A. H., æt. 11 years, was admitted to the Royal Infirmary on 9th May, 1889, on sixty-sixth day of illness, suffering from cerebral abscess of traumatic origin, the injury having been inflicted ten weeks previously.

History.—*Heredity.*—The family history was good, there being no tubercular disease. *Personal.*—Patient had been in perfect health until he was injured.

Present Illness.—Ten weeks previous to admission (28th February, 1889) he was struck on the forehead with a stone, which produced a wound about an inch in length, situated on the front of the brow, an inch above the left eyebrow. It was supposed to be only a scalp wound. Patient was dazed for half an hour after having received the blow. He then appeared to recover, and for several days ran about apparently quite well. The wound, however, suppurated, and was treated by bread poultices, and at the end of the third week the external wound was healed. On the fourth day after injury, he complained of pain in the head, which caused him to remain indoors for four weeks; it then became so intense as to compel him during the subsequent six weeks to remain in bed. The pain was of a dull, heavy character, subject to exacerbations of great intensity, causing him to cry out. During the last six weeks he became very drowsy, and, when not complaining of pain, dozed on. Vomiting was frequent, on an average every second day, though sometimes it occurred several times in the twenty-four hours. About a month prior to admission, after vomiting, he several times sweated profusely (rigors ?). His mother says that he had no shiverings, but that he had "peculiar turns," whether clonic spasm or actual rigors is a question. From the profuse perspiration it was probable they were rigors. His bowels were constipated.

State on Admission.—Patient, who was a fairly nourished boy, lay curled up in bed usually on his left side, his head resting on his hand. He seemed exceedingly drowsy, yet he very frequently cried from pain, and tossed fretfully about in bed. Occasionally he would sit bolt upright, staring around him for a few seconds with knit brows and a hazy, troubled, bewildered look, demanding things which when supplied were thrown by him wearily aside. He would then slide back on his pillow, passing again into his drowsy state. There were periods when he was quiet, the cloud had lifted from his brow, his eyes were brighter, and he was apparently at peace. These were, however, of short duration, his equanimity even in them was easily disturbed, a few simple questions sufficing to ruffle it, and to induce a recurrence of petulancy accompanied by exacerbations of pain.

His pulse was slow and weak, his respirations slow and somewhat irregular, and the temperature about normal. Tactile sensibility and muscular sense were exact. Skin and organic reflexes were normal. There was no ankle clonus, and the patellar reflexes were very slight. The dynamometer showed the grasp of the two hands to be equal (18 kilos). Co-ordinate movements were well performed with both hands and feet.

Special Senses.—Eyesight was fairly good; patient could read large type. Pupils were unequal, the left being somewhat contracted; both reacted to light and accommodation, the left side more slowly than the right. There was doubtful paresis of the left internal rectus. The ophthalmoscope showed double optic neuritis.

Ears.—Both ears were normal. Hearing, taste, and smell were unimpaired.

Intelligence was fair when the boy was free from pain and his attention could be obtained. His memory too was good.

There was a firmly healed cicatrix in front of brow an inch above the left eyebrow, which was still tender. He complained of the left side of the head, and sometimes of the vertex as painful, the pain being increased on percussion.

The other organs were healthy.

Course of Case prior to Operation.—During the seven days succeeding his admission, from the 9th to the 17th May, he vomited occasionally, and frequently had marked irregularity of pulse and breathing. In the daytime he had long intervals of peace, lasting many consecutive hours, when he drank his milk, and seemed happy and contented. It was hard to convince those who saw him only during these blinks of sunshine that he was suffering from an abscess of the brain. At night after dropping soundly asleep, he would start with a low shrill cry, would lie awake for a few minutes complaining of pain in the left temple, and would then doze over. This was many times repeated during the night. Frequently there were quivering of the left eyelids and twitchings on same side of the face. Towards the end of the week his sleep was more disturbed, and he moaned from pain for hours.

On the 17th of May, the symptoms presented a new phase. Early on that morning (1 a.m.) his respiration and pulse were markedly weak and irregular, his restlessness was incessant, the pain in his head greater, and vomiting frequently recurred. About seven in the morning he vomited, after which he lay down quietly as if going asleep, when the nurse observed that his face and lips became extremely pale, and he could not be roused. His pupils were both dilated, and his left leg became rigid. A series of circular movements were made with the left arm which were repeated after an interval. These were not convulsive, however, though they seemed to be automatic. The breathing became very laboured. After remaining in this state for five minutes, patient gave vent to a low shrill cry, and awoke, not being aware of what had happened during the interval. While in this state his respirations were 28 per minute, his pulse 60. The temperature immediately afterwards was 97° F.

The vomiting, restlessness, and prostration continued during the next few hours. At twelve noon he had a fit which lasted twenty minutes. It was preceded by vomiting, and consisted of a series of convulsive movements of both upper limbs, more marked in the right. These continued for four minutes, when both arms became rigid, flexed across the chest, the hands being clenched below the chin. During this period both lower limbs became rigid, but they did not convulse. Patient then had a series of opisthotonic attacks, in the first of which he turned on his left side and became bent like a bow, the whole body being rigid,—pleurosthotonos, *arc en circle laterale.* This lasted three minutes. He then became quite relaxed ; this was followed by two other similar opisthotonic attacks, the patient's body being

CHART H.—TRAUMATIC CEREBRAL ABSCESS.

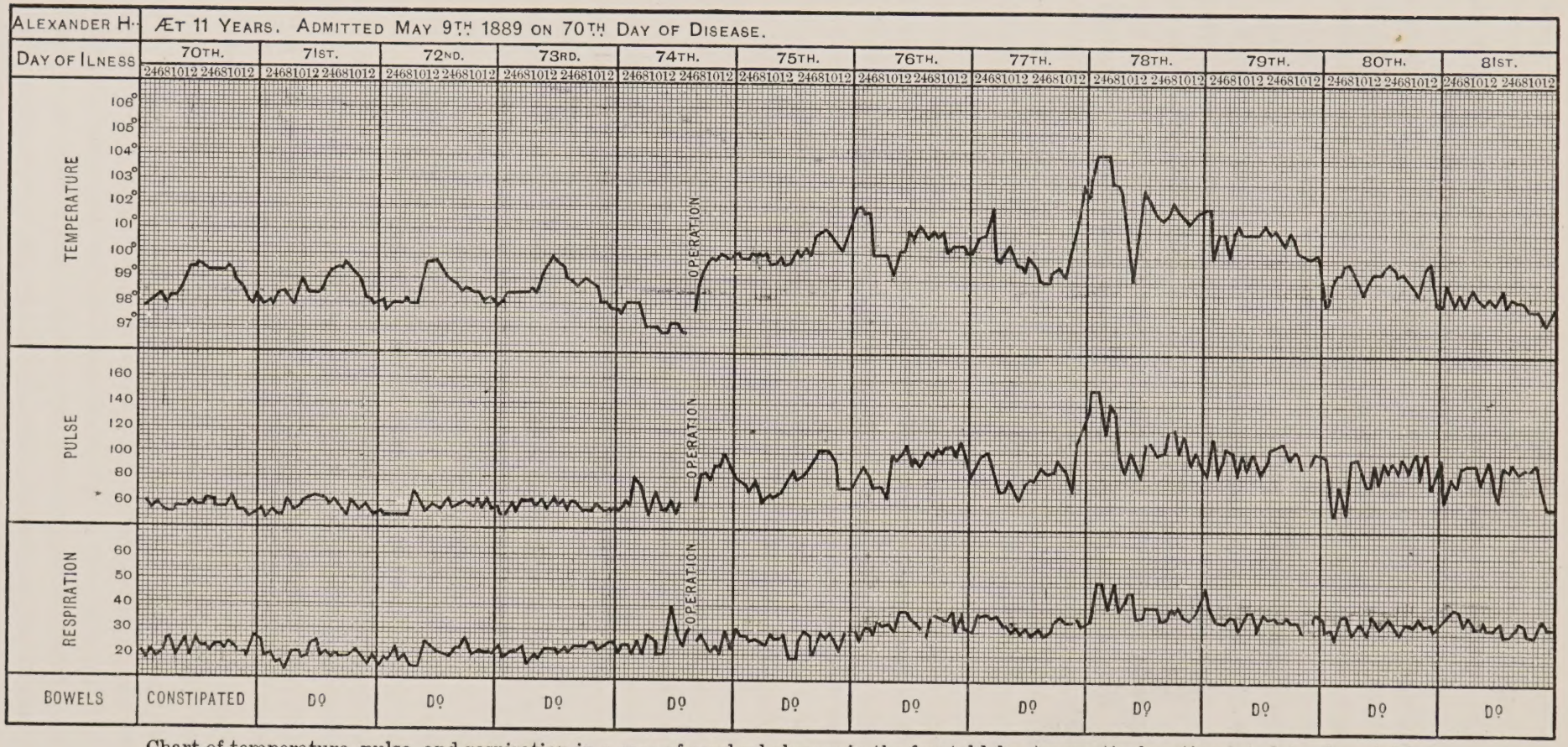


Chart of temperature, pulse, and respiration in a case of cerebral abscess in the frontal lobe, traumatic, bursting into lateral ventricle.

supine, arched, stiff, and resting on his head and heels. The two later attacks were of shorter duration than the first two, lasting about two and a half minutes each. During the fit the pupils were dilated, the left markedly more so than the right. The respirations were stertorous and hurried (38 per minute). There was great lividity of the face throughout. The pulse was very irregular. Three minutes after the fit had passed off, the patient was able to speak intelligently, but was not aware of what had occurred, thus showing his unconsciousness. For three hours subsequently, he lay prostrate in a semi-insensible state and extremely pale.

At 3 p.m. he became totally unconscious, with lividity of face, absolute flaccidity, and absence of reflex movement. His pulse, which had previously ranged from 30 to 60, had risen suddenly to 130; his respiration which was laboured ran about 30 per minute; the temperature was 96°·8 F. The pupils were widely dilated and fixed. This sudden change in the symptoms and their nature betokened involvement of the lateral ventricles. It was hoped that the lining membrane of the ventricles had alone become involved by extension of the inflammatory action, that there had been at most an oozing of pus from the abscess into the ventricles. In either case it was clear that the abscess had extended so near to the anterior horn of the lateral ventricle as to leave only a thin intervening barrier of softened brain tissue. On the other hand, the profound coma, the general convulsions, the opisthotonic attacks, the stertorous breathing, all pointed to the graver view that the pus had actually burst into the ventricles. Immediate operation was decided upon.

Operation (17th May, 3.30 p.m.).—The operation was performed. As he was absolutely comatose, no anæsthetic was employed. An incision was made over the cicatrix, some minute portions of bone were found slightly indented. These were removed and an aperture was made in the skull a quarter of an inch in diameter. The dura mater was thickened and bulged somewhat into the opening, and no pulsations were visible. It was reflected and the inner membranes were found adherent to it, the latter showing some congestion. The brain bulged into the aperture. There was no pus between the membranes or on the brain surface. The surface of the brain seemed rather paler than usual. A hollow needle was introduced into the frontal lobe in a direction backward, downward, and somewhat inwards, so as to strike, if carried far enough, the anterior cornu of the left lateral ventricle. After the brain was penetrated for about an inch a cavity was reached from which pus flowed freely. The pus was of yellow colour, almost odourless, and measured three ounces. With the view of the abscess cavity being more thoroughly drained, an opening was made laterally about half an inch in front of and a little below the stephanion. On this being done the dura mater was found to have collapsed inwards away from the skull towards the abscess cavity, leaving a considerable portion of the interior of the skull bare. This had probably been caused by the removal of the pus. Had the dura been penetrated here, the escaping decomposed pus from the abscess cavity would possibly have collected between the dura mater and the skull, and set up extensive pachy-meningitis. The

dura was therefore left unopened at this point, and the minute drill hole was dressed. A full-sized decalcified chicken bone drainage tube was introduced through the brain by the anterior opening in the skull into the abscess cavity, and a small quantity of carbolized watery solution was injected, but the thorough irrigation of the cavity was not proceeded with as in the other cases, lest the fluid injected would reach the lateral ventricles, carrying along with it particles of decomposed purulent matter, and from possible retention cause over-distention and further brain mischief. The usual wood-wool dressings were applied thickly sprinkled with iodoform.

During the operation pulse and respiration markedly improved after the evacuation of the pus. Sensibility to pain returned towards the end of the operation.

Course subsequent to Operation.—After the operation patient recovered consciousness and slept for some hours quietly. When he awoke, though extremely weak he asked for and drank milk with evident relish. Towards night the restlessness returned, shown by his trying to sit up, turning about from side to side, and making what appeared to be half voluntary, half automatic constant movements of the left arm. Several times there was a doubtful squint of the left eye. The restlessness, the pain in the head, the high temperature continued during the first week subsequent to operation. On the 19th a new feature presented in the shape of diarrhœa, which continued more or less until the 23rd May, then subsiding. At first the motions contained undigested milk, mucus, and blood. The dressings were renewed once during this period, as owing to his restlessness they had shifted somewhat. The wound looked well, though there had been an oozing of pus. On the second dressing three weeks after the operation the drainage tube had disappeared, and the wounds were covered with granulation tissue. They soon healed firmly. Patient was kept in bed for over a month. He was then quite convalescent, and soon recovered his usual health and strength.

Observations of the temperature, pulse, and respiration, taken every hour from the 13th May until the 24th May are here given in chart form. The temperature, pulse, and respiration became normal at the latter date. The optic neuritis began to disappear at the end of the third week, and on examination at the end of July, eleven weeks subsequent to operation, it had all but disappeared.

Remarks.—The history of the period prior to his coming under observation was defective, as the mother, an uneducated woman, could not describe what she saw with sufficient accuracy. There can be little doubt rigors occurred during the initial period, whether observed or overlooked, and it is probable that these 'turns' which the mother referred to were of this nature. The age of the abscess before operation was probably between seven and eight weeks. The symptoms of the involvement of the lateral ventricles became decided on the day succeeding the completion of the eleventh week from date of injury. The amount of pus evacuated (three ounces), if placed in the frontal lobe of a boy, aged eleven, with its anterior boundary an inch from

the anterior surface of the brain, would have its posterior boundary in the immediate proximity of the anterior horn of the lateral ventricle. The symptoms described as occurring on the day of the operation, as well as those manifesting themselves subsequently to the operation, leave little doubt that the lateral ventricles were involved, and that the pus had actually found its way into them. The sudden onset of the grave symptoms, the excessive irritability, incessant tossing about, repeated vomiting, weak pulse and respiration, followed by rigidity of the muscles and convulsive movements, which were bilateral, though most marked on the right side, opisthotonic attacks, and finally the extreme rapidity of pulse, with lividity of the face, extreme prostration, complete coma, and widely dilated fixed pupils, all betokened the bursting of the abscess into the lateral ventricles. A few hours delay in operating would have proved fatal. After the operation, the continuance of the restlessness and semi-delirium, the pain, high temperature, occasional twitching of the muscles, indicated that the irritation had not subsided though the pus had been removed. This persistence of the symptoms of cerebral irritation after the operation was in marked contrast to the conduct of the uncomplicated cases of abscess of the brain in which the ventricles had not become involved. In these the relief of the symptoms was immediate and complete. The state of the brain and its peripheral membranes, as exposed at the operation, showed that there was no trace of external meningitis from bursting of the abscess superficially. In respect of the involvement of the lateral ventricles, this case resembles the second one of cerebral abscess which came under the writer's notice, and which very shortly thereafter manifested symptoms of the abscess having burst into the lateral ventricles. In that case the temperature as well as the pulse and respiration increased with a bound, the coma becoming complete within an hour, breathing stertorous, accompanied by great lividity of the face. The lateral ventricle was found at the operation full of pus, the wall of the abscess on that side having broken down. In contrast to that, in the present case the symptoms though rapid were more gradual, and it is probable that the pus from the abscess had oozed slowly into the ventricles.

CASE XXXIV. *Cerebral abscess, arising from infective traumatism. Diagnosed from the localizing symptoms, to be situated in the immediate vicinity of Broca's lobe. Proposed operation negatived by consultation and friends. Death. Post-mortem operation, trephining and opening cerebral abscess, situated in the white matter near the bases of the second and third frontal convolutions. Remarks. Admitted in third week of his illness. Duration of disease twenty-seven days ten hours.*

In 1876 a boy, eleven years of age, was admitted to the Royal Infirmary, Glasgow, suffering from symptoms which were judged by the writer to indicate cerebral abscess situated in the frontal lobe, in the immediate vicinity of the base of the third frontal convolution.

History.—*Heredity.*—Family history good. *Personal.*—He was a strong active lad until two weeks before his admission, when he fell on to his head from a height, his forehead coming into violent contact with the ground. A wound was caused thereby, of very slight external appearance, and supposed by his friends to be trifling. Little attention was paid to it. He was able to go about apparently well for some days after the accident. On the sixth day he was confined to bed, as he had a headache.

Condition on Admission.—When brought to the hospital on fourteenth day of his illness it was found that he had a partially cicatrized wound about an inch and a half long, situated over the inner part of the left eyebrow. From the centre of this cicatrix a small bud of granulation tissue protruded, and a few drops of fœtid pus could be pressed out. Under the cicatrix a portion of the frontal bone, the size of a shilling, was denuded of periosteum, and felt rough to the probe. He complained of pain all over the front of his head, which was increased on percussion. He was very drowsy, and would drop asleep whenever he was left alone. He had marked want of sustained attention. When roused he would answer intelligently, but in the briefest manner. His rectal temperature ranged from about normal to 100° F. Pulse slow and full from 60 to 65.

His wound was carefully attended to, the granulation tissue removed, the surface of the skull scraped and freely treated with antiseptics.

Six days after admission (the twentieth since the injury) he vomited at nine in the morning. Precisely at the same hour on the following day he had a rigor, which lasted five minutes, after which he again vomited. On the same evening his temperature rose to 103° F. His intelligence was somewhat clouded, and there was a distinct pause after interrogation before he answered a question, his cerebration being slow. His memory was a blank, and his attention could only be sustained for a brief period.

Intracranial suppuration was diagnosed as a probability, its location at this stage being doubtful, but possibly in the immediate vicinity of the wound.

Five days after this—the twelfth from his admission and the twenty-sixth from the date of injury—he had a convulsion, which commenced on the right side of the face, thereafter involving the right arm and right leg, followed by loss of consciousness. The convulsion lasted half an hour, and when it had passed off it was found that the right side was paralysed. When seen by author shortly after the cessation of the convulsion he was distinctly aphasic, being able to utter only the word "No," and when pressed for an answer he would give vent to a series of "No, No," and would shake his head as if annoyed at his inability to express himself. Prior to the convulsion his temperature had reached 105° F.; one hour after it had ceased the temperature had fallen to 98° F. The aphasia lasted one hour, after which he was able to speak and express himself with some little difficulty. He was not so drowsy, and seemed much brighter than he was prior to the convulsion. The right hemiplegia was pronounced for several hours after the convulsion, after which he was able to move his right arm and leg to a slight extent.

From these symptoms a definite diagnosis was formed of abscess in the immediate vicinity of Broca's lobe, situated between the speech centre

and the internal capsule. (See Diagram, Fig. 51.) Had there been a destructive lesion in Broca's lobe, speech would not have been so quickly restored, and the paralysis of the right side would have been more pronounced and of much longer duration. An extradural abscess could cause cerebral pressure, and by inward extension of the inflammatory action so as to involve the soft membranes a lepto-meningitis could occur. In such a case the symptoms would have been more diffuse, and not so precisely limited as in this case.

FIG. 51.—SCHEMA ILLUSTRATING PRODUCTION OF TRANSIENT APHASIA BY AN ABSCESS IN THE WHITE SUBSTANCE OF THE LEFT THIRD FRONTAL CONVOLUTION.

The abscess *A* interrupts only the path *B I.C.* from the speech centre *B* to the left internal capsule *I.C.*, that through the corpus callosum, *C.C.*, remaining unimpaired.

Acting upon the diagnosis of an abscess in the immediate vicinity of Broca's lobe, it was proposed to trephine over the base of the third frontal convolution, and to evacuate the pus from this point. Unfortunately the result of the consultation was to decidedly negative the operation, notwithstanding the proposal of the writer to bear alone the responsibility of performing it. The friends who had been willing to permit the operation while the patient was unconscious, now that he had regained consciousness "and looked so well," wished it deferred, especially as the consultation had negatived the operation. They were told that his improvement would be evanescent and that delay would probably be fatal. The operation was, however, postponed; the parents only giving consent that it might be done were he again becoming unconscious. Nothing further could be done but watch the course of the disease.

For thirty hours subsequently patient's condition remained much the same. At the termination of this period the temperature began to rise gradually till it reached 101·6° F., after which within two hours it increased to 104·8° F. Twitchings then set in on the right side, implicating the parts in the same order as in the first attack. These were followed by convulsions, first right-sided, ultimately becoming general, and lasting until the patient died, about a couple of hours from their onset—twenty-seven days ten hours from beginning of illness.

After death the friends acquiesced in the proposal to have the operation performed just as it would have been during life had permission to do so been granted. From the temporal fossa a disc of bone was removed over the superior part of the base of the third frontal convolution. Between the skull and the dura mater there was a deposit of flaky lymph, after removal of which the dura was seen to be congested. These changes were too slight to have occasioned the symptoms exhibited during life. The dura was therefore turned aside. The cerebral cortex was somewhat congested. A narrow-bladed bistoury was inserted into the brain for fully an inch in the direction of the white matter to the inner side of the upper part

of Broca's lobe. Pus welled out by the side of the instrument, which was left in situ. The calvarium was removed and section was made into the cerebral substance. An abscess the size of a pigeon's egg was found situated in the white matter to the inner side of the third frontal convolution, the blade of the bistoury, which had been inserted through the trephine opening lying in the abscess cavity, having penetrated its outer wall. The tissue in the circumference of the abscess was in a state of red softening, with numerous capillary hæmorrhages. This inflamed zone extended to the wall of the anterior part of the left lateral ventricle in one direction, and nearly to the cortex in the other. The wall of the abscess was flocculent, and numerous portions of brain tissue adhering to the vessels lay loosely in its cavity. There was no limiting membrane. The pus was of a greenish hue and only slightly fœtid. The body was otherwise healthy.

Remarks.—The post-mortem proved the accuracy of the diagnosis, both as to the existence of the cerebral abscess and also as to its exact localization. The localization was arrived at entirely from the data furnished by the symptoms, those of temporary aphasia and post-convulsive right-sided paralysis.

This case furnished an instance of an abscess situated at a distance from the seat of injury in the skull, and *apparently* not arising from distinct cerebral bruising. Had the skull been trephined at the part where the bare bone was situated, the abscess would not have been found directly underneath. It also showed that when symptoms were present pointing to a lesion in one of the recognized cerebral centres, it was better to depend on them as guides, at least to the encephalitis surrounding the abscess, than to trust to the indications afforded by the scalp or skull injury. The presence of such external signs in certain cases is an element of confusion instead of a help. The abscess had arisen from the infective condition of the small external wound which had involved the outer layer of the bone. Pachy-meningitis externa ensued of an infective nature, and the cerebral abscess followed, probably from extension of pathogenic organisms along the course of some minute vessel, possibly by infective thrombosis.

The external wound was small, and the injury to the external table slight. Had this wound been rendered aseptic immediately after production, the abscess would not have formed.

This case illustrates still further the importance of having such patients carefully watched all through by intelligent trained observers. These are required, first, that they may observe the manner in which the convulsions come on, the *protospasm*, as it affords a valuable guide to the localization, which is lost once the patient becomes generally convulsed; secondly, to note the condition of the patient on the cessation of the convulsion, with regard to post-convulsive paralysis and mental peculiarities. Had this patient not been closely observed during the hour subsequent to the convulsion, the aphasic condition would not have been detected. The right hemiplegia soon passed off, only a slight permanent paresis remaining. The careful and frequently repeated observations of temperature also help in these cases.

Here before each convulsion occurred there was a notable and rapid increase, amounting in one case to nearly three degrees within two hours. This sudden rise is often the herald of convulsions, and in any case indicates that changes in the condition of the patient may supervene, requiring the vigilance of the attendants.

Parietal region.—Abscesses in the *parietal region*, which are chiefly due to traumatism, would in all probability affect more or less directly the motor area, and produce very distinctive localizing phenomena, in the shape of convulsions and paralysis.

CASE XXXV. *Cerebral abscess in parietal lobe and suppurative pachy-meningitis subsequent to an infective depressed compound fracture of the skull with penetration of the brain. Trephining, removal of bone from brain, escape of pus, some blood and cerebro-spinal fluid. Washing out of abscess cavity. Re-implantation of bone. Complete recovery. Coming under observation seven days after injury. Duration of illness thirty days.*

D. M'P., aged 70 years, was admitted into the Royal Infirmary on 7th September, 1882, suffering from symptoms of pachy-meningitis and intra-cranial suppuration, following upon a compound fracture of the skull which he had received seven days previously.

History.—*Heredity.*—A long-lived healthy race. *Personal.*—He had been strong and healthy until this injury.

Present Illness.—He had had a shivering on the fifth day after the injury, and when seen on the seventh day he was semi-delirious, very restless, complained of great headache, and frequently vomited. His rectal temperature was 103° F.

Condition on Admission.—On his injuries being examined, it was seen that besides several wounds of the scalp, he had sustained a compound depressed fracture on the right side of the head at the junction of the upper part of the occipital with the parietal bone, close to the longitudinal sinus. The external wound was suppurating, and it was evident from the symptoms that the dura mater and probably also the superficial layer of brain were acutely inflamed and suppurating. He had paresis of the left leg,

Operation.—In order to permit the elevation of the depressed bone and to allow the inflammatory products to escape, trephining was performed. The dura mater was found to be thickened and much congested, its outward surface partly covered with granulation tissue, and between this mass and the skull there were about two drachms of pus. This membrane was turned aside, when it was seen that a portion of the inner table of the skull about an inch in length and three-quarters of an inch in breadth had been driven into and

had penetrated the cerebral substance for about half an inch. Round this fragment of bone there was a cavity containing pus, and on removal of the bone several drachms of foul-smelling pus escaped from the brain substance. After the cavity had been irrigated with an antiseptic solution it appeared red and flocculent, and from its walls several masses of buff-coloured blood clot were removed. Over an ounce of cerebro-spinal fluid flowed slowly away ; it was at first flocculent and turbid, but it ultimately became quite clear. After the wound had been rendered aseptic, the pieces of bone which had been elevated were divided by the chisel and reintroduced, a fear being expressed that owing to the age of the patient and the brittleness of the bone these pieces might not be able to retain their vitality. An antiseptic dressing was applied.

Subsequent Progress.—From the time of the operation he slowly improved, his mind clearing and his temperature falling, and the crural paresis disappeared. The wound healed, all but an aperture through which a fine probe could be passed. This sinus led down to a small fragment of bone, which did not feel loose or distinctly necrosed, and in consequence it was left. After a time the sinus healed firmly, no bone having been shed. He was dismissed well about a couple of months after admission. Two months afterwards he showed himself, when there was a minute sinus leading to an exposed part of bone about the size of a threepenny piece, but quite firm. On digital examination the bone was found to be intact over vault of cranium. There was no hiatus.

Two years afterwards this patient presented himself at a clinical demonstration given to the Southern Medical Society, Glasgow. He was then in good health, and had been regularly engaged at some light out-door occupation since leaving the Infirmary, notwithstanding his advanced age. His head had not troubled him since. The wound on the head was quite healed, and the bone was firm.

Occipital lobes.—Abscess in the *occipital lobes* of the cerebrum is rare except from pyæmia. Pyæmia affects the occipital lobes, the abscess very frequently forming at the seat of infective embolism. An abscess of the posterior aspects of the first temporal, but situated principally in the angular gyrus and occipital convolutions, is reported in Case No. XXXVII.

Cerebellum.—In *abscess* of the *cerebellum* at an early stage there are few localizing symptoms, but when the abscess is large, or when it has set up meningitis of the cerebellar fossa, there is a train of symptoms indicative of involvement of the structures in that region.

In two advanced cases of cerebellar abscess, accompanied by meningeal inflammation, the prominent features were those of extreme depression and prostration. There were retraction of head and neck, extremely slow feeble pulse and respiration, with low

temperature. Yawning, or at least mechanical opening and shutting of the mouth resembling yawning, was very frequent. Along with this there was clenching of the teeth from rigidity of the masseters, with difficulty in voluntary or passive opening of the mouth, and a slow syllabic speech, the words being forced out in a jerky fashion. In both cases there was optic neuritis and blindness, with widely dilated pupils and difficulty or defect in ocular movement. In one there was distinct brachio-crural paralysis with a degree of rigidity, on the same side as the lesion; in the other the degree of prostration was so profound that it was impossible to determine whether there was paralysis or not. The paralysis in such cases may be unilateral and on the same side as the abscess, provided the medulla be affected at a sufficiently low level. It is well to remember that the normal cerebellum passes beyond the foramen magnum and under pressure from abscess it would probably extend further into spinal canal, so that upper part of cord may actually be pressed on. (See Atlas of Head Sections, Plates 36 and 37.) If the pons be most affected on the same side as the lesion then the paralysis will be manifest on the opposite side. If the pressure is general on both sides of the medulla and pons bilateral paralysis would result. The history showed that vertigo and vomiting were marked at an early period in each case. When accompanied by sigmoid sinus thrombosis or phlebitis, there is pain on pressure in upper thirds of both cervical triangles. When abscess bursts into fourth ventricle the temperature, pulse, and respiration are all increased.

An abscess of *the pons*, when small as it frequently is, originating probably in caseating nodules, may present no symptoms until it bursts, when it is apt to induce symptoms of general involvement of structures in the cerebellar fossa. Cases are recorded in which alternate hemiplegia or even bilateral paralysis of the face and limbs was present. Pressure on the pons causing secondary softening may induce ptosis and defect in ocular movement. Diminished sensibility may be expected in a cerebellar abscess compressing the pons. Deglutition is also sometimes affected.

Pressure caused by blood in the cerebellar fossa has given rise to rigidity of the masseters, with frequent yawning of a mechanical kind. In that case, however, the pupils were pin head in size, as if the pons Varolii were affected. Incoordination will likely be found in abscess affecting the middle cerebellar lobe either directly

or indirectly, but the majority of cases which have come under observation have been too deeply affected to permit of incoordination being tested.

In some instances of pressure in the cerebellar fossa, especially implicating the medullary respiratory centre, the respiration was so much affected that it ceased, while the heart continued to beat. This was well seen in a case of hæmorrhage into the cerebellar fossa, in which the heart continued beating for fully five minutes after the last artificial inspiration. In a case of abscess of the cerebellar fossa which was reported to me the inspiration suddenly ceased, the heart meanwhile continuing to work. In this case artificial respiration was maintained for twenty-four hours. During the whole time the heart beat regularly and the circulation was maintained. It is probable, had the pus been allowed to escape, that the respiratory centre would have recovered. No doubt the intrinsic ganglia of the heart contributed here to maintain the cardiac contractions. In cases of purulent cerebro-spinal meningitis affecting the medulla in fourth ventricle, a variety of Cheyne-Stoke respirations occurs, chiefly differing from the ordinary Cheyne-Stoke respiration in the fact that the pause terminates in a long deep breath, instead of a series of very short ones.

In several instances occurring in young children and also in adults the cerebellar abscess and its accompanying meningitis have caused great ventricular distension—a state of internal hydrocephalus. The ventricular distension may be recognized in children during life by the quality of the percussion note, obtainable especially over the pterion. In a case of otitis media purulenta when evidence of abscess exists, if it be indefinite whether the abscess is located in the cerebellum or in the temporo-sphenoidal lobe, the cranial percussion note elicited may aid in the differential diagnosis. The internal hydrocephalus and œdema of the brain may actually be the cause of death in such cases. It is always serious, and when the patient is under the influence of an anæsthetic the respirations must be very carefully looked to as a little increase in œdema may cause a fatal result.

CASE XXXVI. *Cerebellar abscess. Purulent infective otitis media of left side with extensive erosion of the mastoid, perforation of the sigmoid groove and subsequent formation of cerebellar abscess. Operation while in extremis. Removal from cerebellum of from three and a half to four ounces of pus. Complete recovery. Coming under observation at end of three months illness. Duration of illness four months.*

T. K., 17 years of age, was admitted to Royal Infirmary, Glasgow, suffering from cerebellar abscess.

This patient was, on the 14th of May, 1889, removed from a distant part of the country to the Royal Infirmary, but no information concerning his previous illness accompanied him, with the exception that he had been confined to bed for three months; nor was it possible to ascertain the history of his case prior to the operation. This want of history was felt all the more as the patient's state on admission was such that no information regarding the previous course of his illness could be obtained from himself, and there was so much obscurity surrounding his subjective condition that reliance had to be based solely upon the objective phenomena exhibited. These were gleaned during the twelve hours he was under observation prior to the operation. Once during that period he became so pulseless, cold, and collapsed that he was thought by Dr. Waterhouse to be dying, and he was only restored by a liberal supply of external heat and the free administration of brandy.

History.—The history, though placed here in its proper place relatively to the case, was only ascertained by the surgeon some days after the operation, and therefore was wanting when most required. The reader will bear this in mind when considering the position of affairs prior to the operation. As far as can be gathered from the fragmentary manner in which the history was obtained, it is somewhat as follows.

Heredity.—His mother died of apoplexy at the age of forty-three years. Patient was one of a family of twelve, four of whom died in childhood, the others being alive, healthy, and strong.

Personal.—Patient had measles at four. At six he caught cold after over-heating. He was then seized with inflammation in the left ear, causing him great pain, terminating in otorrhœa, and in the formation of an abscess over the left mastoid, which after evacuation, soon healed. The aural discharge continued with variation in quantity since that date. When eleven years old the left mastoid abscess re-formed and discharged pus for some months. Three months ago a third abscess formed over the left mastoid, leaving a sinus, from which a purulent discharge continued to escape on his admission into the Infirmary. He has been deaf on the left side since the time when the discharge first appeared from the ear. During the previous attacks when mastoid abscesses formed, patient did not exhibit any special symptoms, and certainly none referable to the head. The boy was otherwise in good health, went to school, and had a clear intellect.

Present Illness.—Three months before his admission into the hospital, patient returned home from his work one day and went to bed, feeling dull,

and complaining of pain in the back of the head. His left mastoid was swollen. During the first week he lay in a drowsy condition, vomiting when he partook of food, and moaning from pain. The vomiting occurred more or less persistently throughout the remainder of the attack. A week thereafter (third week of illness) he took a "turn," which his medical adviser—Dr. Malcolm M'Lean of Stevenston—stated resembled an "apoplectic fit." He was completely unconscious, with livid, turgid face, stertorous breathing, dilated and fixed pupils, almost imperceptible pulse, and was covered by a cold perspiration. He looked as if about to die. From this state, however, he rallied somewhat, becoming semi-conscious. He was then found to be totally blind, the pupils remaining dilated and insensitive to light. A month from the onset of the illness his left side was observed to be paralysed ; this subsequently improved. Six weeks from the commencement of his illness he lost control over his sphincters. As to his intelligence, he knew the doctor when he heard his voice, and "occasionally passed rather 'graphic remarks' upon what he heard." Toward the latter portion of his illness he had several severe shiverings at intervals of some weeks.

Condition on Admission.—When first seen by the writer in the Royal Infirmary on the morning of the 15th May, his state was as follows :—

On approaching his bed it required some discrimination to assure one's self that the wasted form stretched there was really alive. He lay like a log without sign of animation. Breathing was scarcely perceptible, being markedly slow and shallow, the eyelids were half open and the conjunctiva slightly glazed. The ash grey pallor of the face, with its prominent malars and sunken eyes, the attenuated extremities, the emaciated body, through the shrivelled skin of which the anterior superior iliac spines projected, sores having formed over them as well as on the sacrum, all betokened some prolonged exhausting illness through which patient had passed. He had an extremely feeble, slow, dicrotic pulse, which while he dosed was occasionally imperceptible. His temperature was subnormal. Urine and fœces were passed involuntarily. It was evident that the tide of life was at its lowest ebb.

When roused, which was accomplished with some difficulty, he was conscious. As he was somewhat deaf he had to be addressed in a loud voice. He answered simple questions intelligently, though his replies were profusely intermingled with oaths. His utterance was thick, somewhat muffled, delivered by a series of jerks and in a loud tone, startling as emanating from such a wasted body. Articulation was evidently the result of considerable effort, the masseters being rigid, difficult to move, and at each attempt the face assumed a grimace resembling the *risus Sardonicus.* He did not volunteer information, and his subjective condition could not be ascertained with any degree of certainty owing either to dulness of comprehension or obstinacy in refusing to answer. When asked pointedly as to pain in the head, he occasionally referred to the occiput as its seat. Yawning was a constant feature, occurring every few minutes, frequently several times within the minute. The yawns had a mechanical aspect, and occasionally degenerated into a mere opening and shutting of the mouth. Owing to his extreme

weakness it was impossible to verify any of the lesser degrees of paralysis. He could move both lower limbs and the right arm, the pressure grasp of that hand being about eight kilos. His left arm was rigid, and lay flexed on his chest. It was absolutely paralysed, and the muscles were contracted and wasted. When passively extended and then left free it resumed its flexed position with a jerk as if from spastic muscular contraction. No other muscular paralysis was detected. There was no ankle clonus. The patellar reflex was absent in left, and possibly slightly present in the right.

CHART I.—CEREBELLAR ABSCESS.

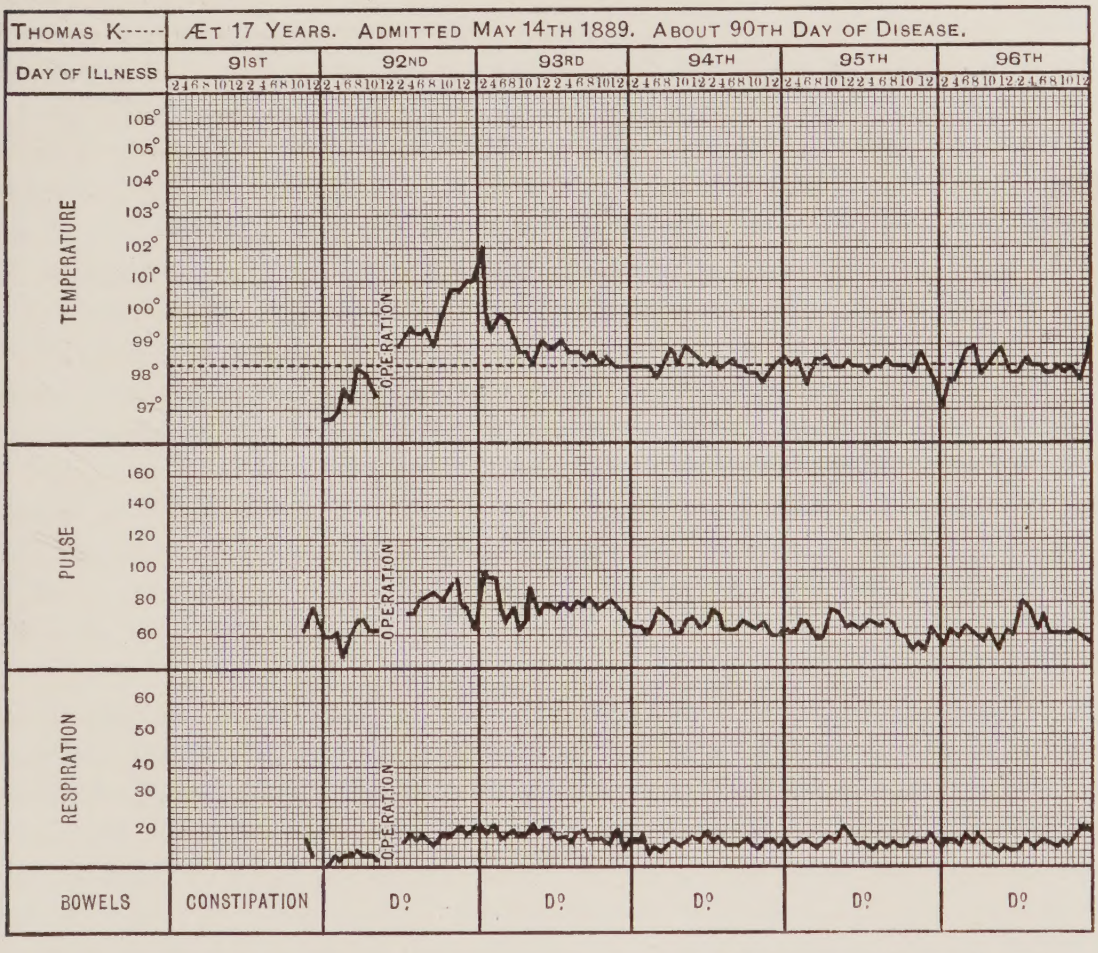


Chart of temperature, pulse, and respiration in a case of cerebellar abscess (uncomplicated).

Cutaneous reflexes were absent. There was considerable rigidity of the masseters, but there was no marked retraction of the head, though the neck muscles were somewhat rigid.

Organs of Special Sense.—Eyesight was gone, the pupils of both eyes were equal and in a state of stabile mydriasis. Voluntary movements of ocular muscles were unimpaired. There was optic neuritis in both eyes, the discs were veiled by whitish grey exudation, the edges being quite indistinct. The exudation occurred in patches along the course of the vessels. The veins were not particularly distended. The arteries were well filled. There were no hæmorrhages.

Ear.—The sense of hearing was extremely dull, or absent in left ear, and was defective in right. The hearing by bone conduction was not tested, as no reliance could be placed on patient's answers, but from subsequent examination after operation it was perfect. The left external ear contained some decomposed pus. The membrana tympani had disappeared, except a small anterior fringe, the space being filled with masses of granulation tissue. There was a minute sinus over the mastoid leading into an osseous aperture from which a drop of pus exuded.

The examination of a small quantity of urine, which was collected, proved it to be non-albuminous. The other organs were normal.

Diagnosis.—The subnormal temperature, the feeble and markedly slow pulse and respiration, the extreme emaciation supervening upon a chronic purulent infective otitis media, pointed in the direction of intracranial abscess, though in the absence of history there were several blanks in the differential signs leading to this conclusion. Some of the objective phenomena might have been attributed to meningitis of the posterior fossa, but there was an absence of retraction of the head and neck so frequently met with in such cases, especially when the meningitis is purulent. Again, the temperature in such is high, the intelligence more obscured, and there are usually indications of general brain involvement.

The rigidity of the masseters, the very frequent yawning, the mechanical opening and shutting of the mouth, the difficult and jerky articulation, the excessive lowering of the circulation and respiration, pointed to a lesion of the cerebellum involving the medulla; while the brachial monoplegia on the same side as the ear affection indicated a lesion below the decussation of the nerve fibres. Blindness is so frequent a sequel of large cerebellar tumours that it also pointed to the location of abscess as cerebellar, and to one probably of large size.

Operation.—The case seemed hopeless, but in intracranial abscess, as in hernia, a patient should not die unrelieved. There was no time to lose. The patient was accordingly placed upon the operating table, when, without anæsthesia, the mastoid antrum was exposed. It was found to be much enlarged by osseous erosions, one of which encroached upon the middle ear, while another had caused a minute defect in the posterior wall of the petrous in the course of the sigmoid groove, through which a bud of granulation tissue protruded. This aperture was large enough to admit the extremity of a probe. The whole of this large cavity in the mastoid was filled with caseated pus and cholesteatomatous matter. It was cleansed, the whole of the eroded bone cleared out, and the remainder rubbed down until it presented smooth walls, upon which any further erosion or lacunar defects would be manifest. The whole was then closely scrutinized, especially the thin wall which separated the middle fossa of the skull from this cavity. The only defect discernible was the one leading into the sigmoid groove.

The external aperture of the mastoid foramen was then exposed. There was no inflammatory indication in it or in the surrounding tissues; there was no oozing of pus from the foramen. When pressure was put on the external occipital vein, the blood from which mingles with that of the vein

issuing from the mastoid foramen, it was seen that the proximal vein, which receives its blood from both these sources, became almost, if not absolutely, collapsed, indicating one of two conditions: a thrombosed sigmoid sinus, or pressure upon this sinus from within the cerebellar fossa, in either case a condition preventing the blood from flowing by the vein issuing through the posterior mastoid foramen. The sigmoid sinus, the greater part of which was covered by granulation tissue, was next exposed for about two inches. Though the sinus was coated by the granulation tissue it was not solid to the touch. There was a small quantity of pus lying between the dura mater and the skull, a drop of which exuded on pressure applied to the granulation tissue. The skull was opened over the left side of the cerebellar fossa, a quarter of an inch behind the sigmoid groove. The dura mater did not bulge markedly out of the aperture, as it so often does in the cerebrum when there is considerable pressure underneath, but it was found to be thickened, vascular, and somewhat softened. While in the act of reflecting it, and just as the inner membranes, which were thickened and adherent to the dura, were exposed, there was a sudden gush of pus, estimated about two ounces, which flowed in a thick stream over patient's neck. When this was cleared away, a wide cannula was introduced into the cerebellum, through which pus to the extent of two measured ounces escaped. A second smaller cannula was introduced at a higher level than the former, and while both remained in the cavity a *gentle* stream of carbolized solution was passed through it; so that the antiseptic solution entered by the narrow upper cannula and escaped by the wider lower one. The washing was done with the greatest care. When completed, a decalcified drainage tube was introduced a little way into the cerebellum, its inner extremity being directed backward. It was stitched securely in position. The wound was dressed in the usual way.

It is interesting to note the immediate effect following the relief of pressure caused by the pent-up pus. This was manifest to all present in the relief to the respiration and pulse, both of which were decidedly fuller and stronger in the first few minutes subsequent to the opening of the abscess. His temperature also participated in the improvement, though this was temporary, as owing to his extreme emaciation it fell to subnormal.

History subsequent to the Operation is one of slow, but uninterrupted recovery, free from complications of any kind. His temperature was often subnormal owing to his excessive weakness and emaciation. During the first twenty-four hours he regained control over his bladder. Within the first forty-eight hours his intelligence had greatly improved.

On the third day he was examined more minutely. It was then seen that his left arm and hand had so far recovered from its paralysed condition that he was able to lift it from the bed, and the power of grasp measured twelve kilos, while he had only double that power in his right. The patellar reflex was present in both knees, apparently to an equal extent. The cremasteric reflex had also returned. The yawning had disappeared as a marked feature, though still too frequent. There was still a trace of the painful expression during articulation, which yet seemed forced. The pupils

were dilated, but reacted slightly to light. In the second week the fundi of the eyes were examined and the contour of the disc was seen to be beginning to appear, the pigmented edge being visible. There was less exudation along the course of the vessels. His eyesight during the first week was very defective, but during the second week it became markedly improved.

At the expiry of the third week the dressings were for the first time removed, when the wound over the cerebellum was found healed. There was still a deep depression into the mastoid cavity which contained granulation tissue. This part and the external ear were refilled with iodoform.

By this period his bed-sores were healed, his nutrition had greatly improved, the sharp angular outline of the bones had disappeared, and his only and constant cry was "more food." Regarding his past illness his memory was a blank; he had no recollection of coming to the Infirmary or of having been operated on, with the exception of having heard a buzzing sound in his ear, this being the noise which the bur used in opening the mastoid makes when driven at high speed.

Six weeks after the operation the following note was taken:—"His intelligence is now quite active, his memory good, his eyesight perfect. The power of his left hand and arm is quite equal to that of the right. He walks about very well, and enjoys life. He has in fact been quite restored." Observations of his pulse, respiration, and temperature had been taken every hour from the time of his admission into the hospital till he was placed on the operating table and resumed after he had been removed therefrom, so that a complete record of these data is here presented. A sphygmographic tracing of the pulse immediately prior to the operation and one obtained just subsequent to it were produced for comparison.

Four weeks after the operation the optic neuritis had greatly diminished, but there were still traces of it eleven weeks subsequently. His mastoid antrum and the cavity leading therefrom to the back of the ear were "papered" with skin, so that there is free access from the middle ear to the outside by this aperture behind the ear.

Remarks.—This case had evidently originated in infective purulent otitis media with subsequent involvement of the mastoid antrum, the walls of which became affected by erosion at an early period. The aperture in the mastoid over the sigmoid sinus gave vent to a somewhat regular flow of pus, small in amount, just such as might be secreted from the granulation tissue surrounding it. There was no unusual discharge of pus from the ear or the mastoid sinus during the three months' illness; it came during that period as a slight oozing, just as it had been coming many years before. It was not connected with the abscess in the cerebellum, which was inside of the membranes and quite shut off from the pus emanating from the granulation tissue outside of the sigmoid sinus. At what time the erosion of the skull into the sigmoid groove occurred is not clear, but it existed in all probability prior to the severe illness which ensued three months before admission. At that time the brain symptoms manifested themselves after the premonitory period usual in intracranial abscess. The peculiar apoplectiform seizure described by patient's own medical attendant might be accounted for in one

of three ways. First, from a somewhat rapid increase in the size of the abscess causing pressure on the important structures in the vicinity ; second, from an involvement of the peripheral or intra-ventricular membranes ; third, from a local hæmorrhage into the abscess cavity arising from ulceration of some vessel under the tentorium, possibly the softened sigmoid sinus. One would have expected to have found some hæmorrhagic vestiges in the shape of debris, minute decolorized clots or in the colour imparted to the pus evacuated from the abscess, but the length of time elapsing from the seizure (ten weeks) until evacuation was sufficient to produce decolorization of the *clot.* Either of the two first explanations is more probable, and it is impossible to decide which is the more correct.

CASE XXXVII. *Case of cerebellar abscess of left side with subdural abscess of large size situated above the tentorium, involving the posterior third of the two upper temporal convolutions, the angular gyrus, and a considerable portion of the adjacent occipital lobe. Word deafness and psychical blindness. Thrombosis of left sigmoid sinus. All arising from acute otitis media following an attack of influenza. Coming under observation fifteen months after attack of influenza. Duration of illness eighteen months.*

J. C., aged 52 years, came under observation in May, 1892, suffering from left cerebellar abscess and subdural abscess situated above the tentorium.

History.—*Heredity.*—Good family history. *Personal.*—Excellent health prior to present illness.

Present Illness.—The particulars as to history of present illness have not been satisfactorily obtained owing to change in patient's medical attendant. The following facts have been ascertained :—In February, 1891, after a severe attack of influenza, he was exposed to cold which caused pain behind the left ear soon extending into the head, involving the vertex and forehead as well as the back of the head. This was followed by loud beating noise and throbbing in the ear, partial deafness, and afterwards by a peculiar numbness over the whole head, and a feeling as if the head were "*squeezed in a vice.*" From the end of March, 1891, he has had a purulent otorrhœa. In May, 1891, an aurist was consulted, who found that the membrana tympani had been perforated. In the following June the pain behind the left ear and back of same side of the head became intense. The tissues at the back of the ear were incised which gave very little relief. From this time he was confined to the house ; the wound at the back of the ear was suppurating and was poulticed. On 13th October, 1891, the parts behind the ear were operated on by an eminent surgeon, for a few weeks after which the pain and discharge were less. Three weeks later the pain increased ; it was especially severe in the forehead and in the back of the head, and the peculiar numb or dead sensation in the head now became persistent. In March, 1892, the same surgeon operated a second time upon

the mastoid region, after which the wound continued to discharge pus copiously, and ten days after he had an attack of erysipelas. From June of 1891 he was quite unfit for business, had little sleep, was very restless, had occasional difficulty in comprehending what was said, and sometimes saw things "not as they ought to be." On two occasions in May, 1892, he had rigors with a week's interval between each.

Condition when First Observed.—When seen by the writer in consultation with Dr. Smith, of Dumfries, in May, 1892, the patient had a vertical granulating wound over the mastoid, from which a large amount of pus emanated, partly from the visible granulation tissue, but chiefly from a sinus in the soft tissues leading to an abscess cavity an inch posterior to the wound. The quantity of discharge seemed greater than could be accounted for by an emanation from the superficial parts. Patient, who had a peculiar greyish colour of face, complained of great pain over the back of the head and in the forehead. He had no motor or sensory paralysis, though he stated he was weaker than he had been prior to his illness. There was slight optic neuritis in both fundi, the right being the worse. In the left external meatus there was some purulent discharge, the membrane had been perforated, and there was a small bud of granulation tissue protruding from the middle ear through the membrane. His memory was defective, especially for recent events.

During the first few days after coming under observation he frequently answered questions in such a way as to cause one to believe he had not heard what was asked. On closer observation it was apparent that his hearing on the affected side was good, he could hear ordinary conversation on that side, but he did not seem to understand the words spoken. His answers were in themselves coherent and correct statements though they were not *à propos* of the question. At first he sometimes became aware that his answers had not been what was expected by watching the facial expression of his audience; when he found it indicating surprise, he would at once stop short, and ask the question to be repeated slowly to give him time to grasp its meaning. Occasionally the question had to be repeated several times before he gave the right answer, and it seemed to have been arrived at on his part by a process of guessing or surmise as to what the question probably was. He resembled a foreigner trying to understand a language with which he had little acquaintance, with this difference, that whenever he wished to express himself he had complete command of the language and talked coherently and intelligently. His condition raised the question whether he was suffering from a degree of "word deafness," which became highly probable when it was found that he was deaf in the sound ear, though he could hear very well with the affected ear, and the deafness on the sound side had been present to an extent for a couple of months, though it had only become pronounced within the last few weeks. The deafness was sensory, and the difficulty about answering questions was due to a defect in the auditory perceptive centre.

The other symptoms and appearances pointed to a lesion in the cerebellar fossa, probably meningeal, but possibly also with small abscess. It was

difficult to understand what connection, if any, the lesion in the posterior third of the first temporal had with that in the cerebellum, and it was possible that there were two independent lesions.

Operation.—The mastoid region was exposed to ascertain its condition. An opening in the sigmoid groove was found filled with granulation tissue. This opening was evidently artificial, and had probably been made by former surgical operations. There was a sinus in the soft parts leading to the posterior margin of the mastoid over which a small abscess was situated.

It was then resolved, first, to open the mastoid antrum, clear out its contents, and to examine the tegmen antri and tympani; and, secondly, to open the cerebellar fossa. The antrum mastoideum was situated half an inch in front and the same distance deeper (nearer the middle line) than the opening which had previously been made into the knee of the sigmoid groove. It contained pus and granulation tissue, as did likewise the middle ear. After removal of the debris and rendering part aseptic, the tegmen was carefully inspected, but no erosion was detected. The sinus which led into the small abscess at the back of the mastoid was opened, and showed that the soft tissues over the mastoid were deeply stained of a dark colour. The putrid pus, granulation tissue, and small sloughs were removed, and the cavity washed out. Opening into the cerebellar fossa was postponed, first, as patient had been under chloroform for a prolonged period; and, secondly, to ascertain whether pus from the interior of the skull found a vent through the posterior portion of the mastoid at the seat of the old abscess, or whether it issued by the aperture in the sigmoid groove. It was seen at the subsequent dressings that pus issued from both, but the greater quantity was from the posterior aspect of the mastoid. It was difficult to find an entrance into the cerebellar fossa at that part, though it was clear that one existed. The position of the mastoid foramen was exposed, and though the tissues round its orifice were œdematous, there was no pus issuing from it. The vein, however, seemed collapsed, and was smaller than usual. A few days after the first examination and opening of the mastoid antrum, the cerebellar fossa was opened behind the mastoid, and, at the same time, the opening into the sigmoid groove was considerably enlarged. At both points pus was found outside the dura, and the latter was thickly covered with granulation tissue, which extended over the greater part of the lateral aspect of the cerebellar fossa. After careful washing of the granulation surface, pus continued to ooze out, especially when pressure was exerted on the granulation mass, which covered the dura. The latter was therefore incised, and inside of the cerebellum an abscess containing about half an ounce of pus was found. Its cavity was walled off from the surrounding tissue, and it was therefore chronic. This cavity and the corresponding cerebellar fossa was well washed, the fluid entering by the posterior opening, and gaining an exit by the sigmoid groove. The sigmoid sinus was thrombosed, but the thrombus was not disintegrating.

It was distinctly disappointing to find such a small abscess in the cerebellum, and highly improbable that the amount of pus found daily on the dressings previous to this opening of the cerebellar abscess could have

emanated from such a small area. The possibility of another abscess reaching and affecting the posterior aspects of the first temporal convolution became an important question, though, if it did exist in that area, it was considered improbable that any pus from the cerebrum would find its way through the tentorium, and escape by an opening in the skull below the lateral sinus. An opening in the bone below, but passing obliquely upward toward the parieto-squamo-mastoid suture was discovered, through which an occasional drop of pus escaped. A fine probe introduced into this opening passed in an upward direction toward the posterior part of the cerebrum, and while passing the probe about a drachm of pus escaped by the cerebellar trephine opening.

It was resolved to delay opening the cerebral fossa, in order to see the result of the evacuation of the cerebellar abscess.

His symptoms, however, became worse. He became absolutely "word deaf," and could not understand anything that was said to him. While in this state, his attitude appeared to some of his attendants like that of an "obstinate man" who would persist in having his own way, without listening "to reason"; who, though reasoned with, would answer irrelevantly and pursue his own course. Next day, in addition, another symptom appeared. He seemed as if he did not see or could not understand what he saw. When he wished a drink, the vessel from which he usually received milk was placed right in front of him in line with his eyes, but he did not recognise it. All endeavours to attract his eyesight to the object failed. Sometimes, however, he saw that *an object* was presented to him, and he would ask what it was. When his lips were touched with the spout of the drinking cup, he at once tried to drink, and his hands endeavoured to catch the vessel. Again, if a vessel of another shape than that from which he usually drank was given him, it was evident he did not recognise by eyesight the altered form. When a tumbler was held to his lips—he being usually fed from a drinking cup with a handle—his hand at once went up and endeavoured to catch a supposed handle on the tumbler, in the same way as one would catch the handle of a tea cup. As soon as he grasped the vessel, he quickly recognised its form through the sense of touch. He could therefore understand neither what he heard nor saw.

There was now sufficient evidence to warrant opening of the cerebral fossa, to expose the posterior aspect of the first temporal and the angular gyrus. This was done by removing a disc of bone over the first of these positions, when a wide cavity was exposed capable of containing over three ounces of fluid, and extending from the region of the first and second temporal convolutions at their posterior aspects backward and downward over the occipital lobe as far as the tentorium. A second trephine opening was made over its posterior limit to afford complete drainage. It was seen that the probe introduced from the cerebellar trephine opening reached this wide cavity over the cerebrum, and the foramen through the parieto-squamo mastoid suture also afforded entrance into the same cavity. This large cavity was lined on all sides by a thick, dark, soft pulpy membrane, which was continuous over parietal and visceral sides. It contained about three

ounces of pus, which partly escaped by the lower openings and partly by the trephine openings now made. After the cavity was washed nothing could be seen of the relation of parts through this thick dark membrane. The dura seemed to line the bone in its normal position. It was, however, difficult to differentiate a separate dura mater from this pulpy membrane, which was adherent to the parietal side of the cavity. On the visceral side the membrane was equally thick, and after cutting through it, the brain for the first time was exposed and its pulsations felt. The cavity was in the subdural space, and must have existed for at least months prior to opening. It was now clear that the foramen or erosion in the skull gave vent to the pus from this cavity, and that the cerebellar fossa communicated with the cerebral fossa, the tentorium being at one part eroded to permit of this. The brain had been so much and for so long compressed that it showed no signs during the operation of regaining its former relation with the bone. This, however, it was hoped would take place gradually.

The result of this operation was highly satisfactory. Within four days he regained the power of understanding what he saw, and this was followed four days later by clear indications of returning comprehension of spoken words. At the termination of three weeks from the date of this last operation, he was perfectly clear in both these respects, and, though weak, regained strength daily. The deafness on the sound side had likewise disappeared, and at the same date the hiatus within the posterior segment of the skull had become obliterated by the expansion of the brain. He progressed rapidly in strength from this time. His understanding was perfect, and he had no mental defect. His wounds healed by granulation tissue. On bacteriological examination of the pus which escaped from the cavity in the occipital lobe Frænkel's pneumococcus was found in abundance along with the staphylococcus aureus.

CASE XXXVIII. *Cerebellar abscess with sigmoid sinus thrombosis arising from infective otitis media. Operation. Recovery. Coming under observation in seventh week of illness. Duration of illness ten weeks.*

T. C., 29 years, a labourer, was admitted into Royal Infirmary in 1891, suffering from infective otitis media, with symptoms of thrombosis of sigmoid sinus and cerebellar disturbance.

History.—*Heredity.*—Family history indefinite. *Personal.*—Patient had been a healthy man, who never had any illness except such as arose from injury. Ten years previously he had an escape of pus from his right ear, which lasted about a fortnight. It then ceased. Since then he has never had any discharge from the ears until his present illness.

Present Illness.—Nine weeks ago he caught cold, having been working under water as a diver for a considerable time. A discharge appeared from his right ear, accompanied by a severe pain in both ear and head, which confined him to the house for five days. He subsequently returned to work, but a fortnight afterwards was again attacked with severe pain in the right ear and head, accompanied by swelling and tenderness to touch behind the

ear. During this time he had on three occasions a sensation of cold which amounted to a rigor. He also experienced considerable giddiness, especially when he stooped.

Condition on Admission.—There was slight œdematous swelling and redness over mastoid, extending two inches backward from the pinna, also swelling and tenderness along the whole course of internal jugular, especially marked at its upper part. The membrana tympani was perforated and blocked by granulation tissue. A watch was heard at six inches on affected side, and at four feet on sound side. No ocular symptoms, though squint was stated to have been present previously. He was rather excited mentally, had marked vertigo when he stood erect, and an unsteadiness of gait. He had a temperature on admission of 101·4° F., and rather a slow full pulse. He yawned frequently, and his masseters were rigid. His head was retracted, but he could bend it forward by a painful effort.

Operation.—The mastoid antrum, middle ear, and mastoid cells were freely exposed, and found to be filled with fœtid pus and granulation tissue. There was a large erosion in sigmoid groove, and masses of granulation tissue surrounded the sinus. The anterior wall of the sinus consisted of granulation tissue, which on removal exposed a thrombus in process of disintegration. On removal of a portion of this thrombus, the posterior and inner wall of the sinus was found softened sufficiently to admit a probe into the cerebellum. On this being done, a few drops of pus oozed out. On the softened part being enlarged, and the cerebellum exposed, about three drachms of pus escaped along with a small portion of brain substance.

The cavity of the middle ear was cleared out, two of the ossicles (malleus and incus) being removed. The malleus, which exhibited erosion, was found embedded in a mass of granulation tissue. The sinus was opened up for fully one inch, the disintegrating debris removed, the space filled with iodoform and boracic acid, and the whole cavity of the mastoid and middle ear was filled with the same.

Subsequent Course.—Patient pursued an uninterrupted course towards recovery, with the exception that during the first fortnight the pulse and temperature were subnormal, the former markedly slow and full, sometimes being about forty per minute. The aperture into the mastoid was "papered" with skin.

Remarks.—Though the history of this case makes it appear as if it were acute, it is probable that some very slow disintegration had been going on unobserved in the middle ear and neighbouring parts. The fresh access of inflammation occasioned in the middle ear new activity, which had extended to the cerebellum. The symptoms pointed to involvement of the cerebellum along with thrombosis of the sinus, the slow pulse rather indicating abscess, while the high temperature and retraction of the head were indicative of slight meningitis of base, such as might arise from thrombosis of the sinus. The pain along the course of the internal jugular pointed to phlebitis.

This patient presented himself several times to report. He was last seen in July, 1892, in excellent health, and quite fit for work and regularly at it. There was no discharge from his ear.

CASE XXXIX. *Case of cerebellar abscess with partial thrombosis of sigmoid sinus, arising from infective otitis media. Operation. Recovery. Coming under observation on the sixtieth day of illness. Duration of illness ninety days.*

J. R., aged 38 years, was seen in consultation with Drs. Wood Smith and Barr, on 13th February, 1892, suffering from cerebellar abscess, with partial thrombosis of sigmoid sinus.

History.—*Heredity.*—Family history good. *Personal.*—Patient has had left facial paralysis and deafness since early childhood, but for many years there has been no suppuration in the left ear. Otherwise he has been in good health.

Present Illness.—On 15th December, 1891, he got chilled; felt pain in the right ear. Six hours afterwards he awoke in great agony with pain in the right ear, shooting thence through his head. Some days later the pain had further extended to the vertex and back of head and right side of neck. Along with the pain he had deafness in that ear. After a fortnight's treatment in bed he felt stronger, but the pain and deafness persisted. He was however able to be taken to the country for a change of air, from which he benefited, but was never free from pain in the head and back of right side of neck. He returned from the country on the 11th February, and the same night and next day the pains becoming much aggravated, the writer was asked to see him on 13th February.

Condition when First Observed.—He then complained of pain over right ear, vertex, and more especially over the back of head and right side of neck, and he had pain on pressure over the course of the upper third of the jugular vein and at the apex of the posterior cervical triangle, in both of which regions the tissues were thickened and contained several enlarged deep cervical glands. There were several small superficial submastoid glands likewise enlarged and painful to touch. The pain he described as persistent and throbbing.

The motor power (with exception of left facial paralysis), sensations, and reflexes, were normal.

A slight purulent discharge emanated from the right ear, and the membrana tympani had a wash-leather appearance. There was slight papillitis in both fundi, especially the right. The pulse was 55 per minute, and the temperature was 99° F. His lungs were free from evidence of infective invasion. These symptoms pointed toward thrombosis of the sigmoid sinus with cerebellar suppuration, and immediate operation was advised.

Operation (13th Feb., 1892).—The mastoid antrum was opened and found to contain granulation tissue and pus. The sigmoid groove was then exposed. Masses of granulation tissue, sprouting from the knee of the sigmoid sinus, projected through the groove into the mastoid cells. These granulation masses being removed, the dura of the cerebellar fossa was found thinned at the inner and anterior side of the sigmoid sinus, and a drop of pus exuded by the inner side of the sinus through a minute erosion in the cerebellar dura. The dura was opened at this part, and this gave vent to about three

drachms of pus, which issued from the cerebellar tissue, which was at that part softened. The sigmoid sinus, though distinctly thickened, was not opened, as the mass of granulation tissue surrounding it in all probability had acted as a protection against the entrance of infective organisms. The cavity was washed out with a weak carbolized solution, the antrum as well as the middle ear being cleansed. The ossicles and membrane were left intact.

From that time the pain previously experienced greatly diminished, and he was soon able to have refreshing sleeps and to awake feeling well. The pulse still continued slower than normal, 50 to 55, though his normal pulse and that of some other members of the family was about 60. His temperature was normal. At the termination of a week he was quite free from pain. The tumefaction of the tissues at the upper part of the neck had almost disappeared. A fortnight afterwards the enlarged glands had greatly subsided. The wound closed by granulation tissue from below upwards. He made an uninterrupted recovery and regained his former health and strength.

CASE XL. *Cerebellar abscess complicated with diffuse internal and external suppurative cerebro-spinal lepto-meningitis due to left otitis media. Not operated on. Death. Coming under observation a month after commencement of illness.*

G. A., aged 19, riveter, admitted into Royal Infirmary (in the absence of Dr. Macewen) on 14th August, 1890, suffering from cerebellar abscess, complicated with diffuse internal and external suppurative cerebro-spinal lepto-meningitis, due to left otitis media.

History.—*Heredity.*—Obscure. *Personal.*—Patient, since he was seven years of age, had a suppurating discharge from the left ear, which came on after an attack of measles. He had been rather neglected as a boy, and was of under average intelligence. When about sixteen years of age (three years ago) he had a mastoid abscess which burst at the back of the ear. A cicatrix now marks the seat of the abscess.

Present Illness.—Information was very scanty and defective. About a month ago he had an attack of vomiting, dizziness, and pain in the head, which lasted for a few days. Afterwards he remained well till six days ago, when he became affected with severe pain behind the left ear and in the forehead. The pain was so severe as to prevent him from rising from bed, and it was accompanied by giddiness. He vomited the same morning. During the subsequent five days the pain persisted in the head. Although the other symptoms were allayed, the pain in the ear continued.

There was no history of rigors.

Condition on Admission.—Patient was small and poorly developed, markedly rachitic and ill-nourished. He complained of pain in the head, chiefly over the left mastoid and frontal regions, not increased on tapping.

There was an old cicatrix over the left mastoid where an abscess had burst three years ago. No swelling or thickening about the jugular vein. Muscular system unaffected by any paralysis. He toed a line, stood steadily with closed eyes, knee jerks were a little increased but equal, the plantar reflexes were active, slight ankle clonus. Ocular muscles acted normally, pupils equal, medium, reacted to light and accommodation. Sensations were normal.

Fundi normal when admitted, hypermetropic astigmatism, outline of discs clear.

Hearing.—Did not hear a watch at all on the left side, on the right only at six inches. The left membrane was not seen owing to a mass of granulation tissue which sprung from the posterior wall. The right membrane was indrawn.

Temperature on admission 101·4° F., on the subsequent days temperature ranged from 99°·2—103·8°. On the 17th of August there was an extensive crop of herpes facialis, involving lips and distribution of nasal and infra-orbital nerves on left side.

18th August (evening).—Distinct optic neuritis in left eye. Right as before.

Operation (19th August, 1890).—The mastoid cells were opened as patient had become decidedly worse.

Subsequent Course.—The right pupil became dilated and the right eye inflamed, and there was slight left facial paralysis. On the subsequent days in addition to the foregoing there was pain in the chest, especially on the left side, and pain in the forehead and neck, passing down the neck and back. There was slight retraction of the head and marked rigidity of the back, and somewhat later there was profuse perspiration, followed on the 21st by delirium and unconsciousness.

He had a slight cough and sighed frequently, and towards the end there was squint, both eyes being turned to the left.

As the fatal termination, which occurred on the 24th, ten days after his admission, approached, his temperature ran up from 104° to 108° F.

Post-Mortem Examination.—Showed extensive suppurative lepto-meningitis, both middle and posterior fossæ being lined with pus, which extended the whole length of the cord. On the left side of the cerebellum there was a considerable quantity of pus which had escaped from a ruptured cerebellar abscess in the left hemisphere, the rupture having taken place in front and towards the outer side of the cerebellum, in close proximity to the sigmoid sinus.

On section of the brain all the ventricles were found greatly inflamed and contained a quantity of pus. (The position and appearances of the cerebellar abscess and ventricles are seen in the photograph.)

There were recent adhesions between the pleura and lung on both sides of the chest: no effusion on either side. The base of right lung was a little congested, the left lung being more so, and œdematous.

CASE XLI. *Cerebellar abscess complicated with lepto-meningitis—purulent in the cerebellar fossa, serous in the cerebral fossa and round cord—and internal hydrocephalus. Death, without operation. Admitted on thirteenth or fourteenth day of last acute illness—but probably six months after first formation of abscess. Duration of abscess probably about seven months.*

M. S., female, aged $4\frac{8}{12}$, was admitted into the Children's Hospital, Glasgow, on 15th December, 1890, affected with cerebellar abscess, complicated by cerebro-spinal lepto-meningitis and internal hydrocephalus.

History.—*Heredity.*—No hereditary tendency to tubercle. Parents have three other children, all healthy. *Personal.*—Patient had been in good health, with the exception of a slight attack of measles and a severe one of scarlet fever, with sequelae. For the latter ailment, she was admitted to Belvidere Fever Hospital in May, 1890. Dr. Carslaw, under whose care she was while in Belvidere, has kindly supplied notes of her condition while in that hospital, from which the following is an abridgment.

The case was one of severe scarlet fever. On the twenty-first day of illness, left otorrhœa appeared. On the thirty-fifth day, nervous symptoms showed themselves with amaurosis and stabile mydriasis.

On thirty-eighth day an abscess which had formed behind the left ear burst, and a quantity of fœtid pus escaped.

On fifty-fourth day a probe passed through the sinus led to bare bone.

On the sixty-ninth day the child could see again, and there was general improvement. After leaving Belvidere Hospital, the sinus behind the ear which had closed reopened, and from that time continued regularly to discharge fœtid pus. There was no pus from the ear after she left the hospital. Otherwise she continued very well for three months, always able to go about.

Present Illness.—On 2nd December, 1890, she complained of pain in the forehead, preferred to be in bed, and had no appetite. No shivering was observed by parents.

On 4th December, the headache persisted, she vomited twice, her speech became thick, and the articulation difficult. There was also slight retraction of the head.

On 11th December, ninth day of present illness, she had become gradually worse, the headache severe, the vomiting recurring on any attempt to assume the upright position. She often uttered a sharp cry (hydrocephalic ?) while asleep. The bowels were constipated. About mid-day on this date a fit occurred, marked by the following:—deep sighing, extreme pallor of face, turned up eyes, opisthotonos, rigidity of arms and legs, slight flexion and full pronation of arms, with flexion of fingers and thumbs. This condition lasted two minutes, after which she fell asleep for a quarter of an hour, then became conscious. At

midnight a second and somewhat similar "fit" came on and lasted twenty minutes.

From this time, till date of admission, these fits continued to recur with gradually shortening intervals, and during the last few days the child became blind, though this fact was rather difficult to determine owing to her semi-conscious state.

State on Admission.—During the first forty-eight hours.—On the thirteenth and fourteenth day of the disease her condition was as follows. There was marked retraction of the head and arching of the back, which occasionally became extreme. When this somewhat subsided, the abdomen became shrunken and retracted as one sees in cases of lepto-meningitis of a tubercular nature in their last stage. The face was pale, though it occasionally flushed. The pupils were widely dilated and fixed, the eyes being amaurotic. The eyes, as well as the head, were slightly turned to the right. There was double optic neuritis, the left fundus being worse. The left membrana tympani had disappeared, the ossicles could be easily seen. The membrane of the right was intact, but in the external auditory canal there were purulent granulations. Fœtid pus issued from a sinus over the left mastoid which entered the sigmoid groove, a small quantity of pus escaping on each introduction of the probe, evidently coming from cerebellar fossa. Opening and shutting of the mouth as in yawning was frequent. There was great difficulty in swallowing even when patient was semi-conscious. She frequently sank into a comatose condition from which she could not be roused, and during which she frequently uttered sharp screams (hydrocephalic ?).

Occasionally, she became semi-conscious, and cried with pain in front of head. Her speech was then slow, syllabic, and drawling. There was no apparent paralysis, but the feebleness was so great, and was so coupled with her semi-insensible state that it was difficult to determine this.

Patellar tendon reflexes were slightly exaggerated, no ankle clonus, and no anæsthesia of limbs, but this was doubtful. Percussion over vertex and pterion gave hollow note indicative of over-distension of ventricles. The pulse was very feeble, scarcely perceptible, and the temperature was sub-normal. She died on the fifteenth day of her second illness, after being under observation for two days.

Post-Mortem Examination.—The spinal cord was first exposed, when there was found to be marked lepto-meningitis with large amount of serum within the membranes. There were fibrinous flakes in the fluid but no pus.

The cerebellum was exposed from above. The inner membranes were congested and purulent, especially the left, which were also in parts disintegrated. On its left hemisphere and between it and the dura, pus was found, communicating with an abscess in the left lobe of cerebellum which extended into the pons. The abscess in the pons had firm walls, which were easily detected through the substance of the cerebellum, as they were of the consistence of a caseating nodule. There was an erosion in the dura mater establishing communication between the cerebellar abscess and the exterior of the skull. The pus which remained was greenish,

fœtid, and measured half an ounce, but it was evident that had it not been for the escape of the pus through the channel, the quantity would have been much larger.

The cerebrum had its pia mater and arachnoid fused and both were deeply congested, milky, and turbid. The convolutions were soldered; there were no distinct tubercles. The ventricles were enormously distended with chronic serous effusion. Both lateral and the third were greatly distended, and even the fifth was involved in the distension. The third was somewhat circular and measured half an inch in diameter. This ventricular distension accounted for the clear note given on percussion over the pterion and vertex generally.

The petrous portion of temporal bone.—The erosion passed through the mastoid portion of the petrous into the groove of the sigmoid sinus. The sinus was obliterated by previous thrombic action, and at the anterior aspect of the sigmoid the dura was perforated, a bud of granulation tissue filling the orifice and projecting into the groove. A second and larger perforation was found anterior to the former, situated at the knee of the sigmoid groove. This communicated with the tympanic antrum. This second perforation was filled on the side of the cerebellar fossa with masses of granulation tissue (see Fig. 35).

Remarks.—It is probable that the latter erosion described had been the first formed, and through it the cerebellar fossa had been invaded, the sigmoid thrombosed, and an extradural abscess formed, followed by one in the cerebellar hemisphere. The erosion through the sigmoid groove to the exterior of the skull was secondary, but after its formation the pus escaped by this direct channel instead of through the tortuous, more anterior, and higher antral one. The cerebellar abscess had in all probability formed after the scarlet fever about seven months previous to her death. When in Belvidere, the head symptoms and blindness were probably due to the formation of the abscess, and the relief experienced followed the leakage of the abscess externally. The subsequent attack commencing in December was evidently meningeal, and symptoms described were then due both to the cerebellar abscess and the cerebro-spinal lepto-meningitis. The child was admitted in a dying state, and from the complications presented and the prostration it was deemed inadvisable to operate.

CASE XLII. *Cerebellar abscess with basilar meningitis of posterior fossa, originating in chronic otitis media, the pus of which had become infected with erysipelatous organisms. Coming under observation on third day of illness. Duration of illness sixteen days.*

The following précis is taken from the notes of this case kindly supplied along with the fresh specimen of the brain, its membranes, and the temporal bone, by the gentleman under whose care it was. The description of the pathological appearances is by the author.

W. D., 11 years, school boy, was admitted under the care of Dr. F. on 7th December, 1891, as a case of erysipelas.

History.—*Heredity.*—No family peculiarity. *Personal.*—He had always been in good health with the exception of having had a purulent discharge from the right ear, which had lasted for ten years, and which had gradually increased in quantity.

Present Illness commenced on 4th December, 1891, when he complained of great pain in the right ear, the purulent discharge at the same time increasing. On the second day of his illness there was an erysipelatous blush over the right side of his face, and the right eye was closed by the accompanying swelling. On the third day of illness, when admitted into hospital, the whole of the right side of his face and head was œdematous, the right eye was closed, and there was a profuse fœtid discharge from the right ear. His temperature was 101·2° to 102° F., and he was mentally dull and drowsy.

On the fifth day the erysipelas had disappeared. His temperature was normal. He had convergent strabismus, and pressure over the mastoid caused acute pain. On the sixth day of his illness an incision was made in the mastoid region, which gave vent to several ounces of fœtid pus. By this escape of pus his condition improved for eight days, after which there was an increase in his symptoms, and on exploring the wound with a probe a further quantity of fœtid pus escaped, which again relieved him. The patient, however, gradually sank and died on twenty-first day of his illness. His eyes had been examined by ophthalmoscope, and slight papillitis was discovered.

Examination of Brain and Portion of Skull.—The temporal bone as examined by writer showed enlargement of middle ear and antrum at the expense of their walls, which were affected by ulcerative caries penetrating the sigmoid groove. The whole of the interior of those parts was blackened, more especially over the tegmen tympani (which was also eroded) and the thin plate of the mastoid over the sigmoid groove. Both malleus and incus were involved in the ulceration, and both were of a very dark colour. The floor of the middle fossa presented a dark appearance along the line of the petro-squamosal suture, and over the tegmen generally. The dura covering these bony parts, and for half an inch beyond them, was inflamed. There was a series of small vessels running from the bone into the superior petrosal and sigmoid sinuses, as well as some cerebral vessels over the base of the temporo-sphenoidal lobe, of which all were thrombosed. In the sigmoid groove at the knee of the sigmoid sinus the bone was very dark, and dotted with minute spots of greater density of colour, which were the orifices of thrombosed vessels issuing from the bone. At the upper part of the sigmoid groove there was an aperture caused by erosion communicating with the mastoid antrum, and through this aperture a mass of granulation tissue protruded from the sigmoid sinus. There was a second aperture, evidently artificial, from the exterior of the bone into the sigmoid groove. These two apertures did not communicate with one another.

The dura was inflamed, thickened, partly covered with granulation tissue, and separated from the bone opposite a part of the sigmoid groove, and for a considerable distance over the posterior aspect of the petrous bone. There was an erosion in the dura to the inner and anterior aspect of the sigmoid sinus through which the pus in the cerebellar fossa escaped. The sigmoid sinus was thrombosed, the thrombus being of old standing.

The brain showed evidence of extensive serous lepto-meningitis, while over the base from the pons backward into the cerebellar fossa, and especially on the right side, there was suppurative lepto-meningitis. In the right lobe of the cerebellum there was an encysted abscess cavity about the size of a large walnut, thinned and ragged anteriorly, and communicating with the rent in the dura to the inner and anterior aspect of the sinus. The upper part only of the spinal cord was included in the specimen, and its membranes were covered with pus. (See woodcuts 43 and 44.)

Remarks.—The history of nine years' duration of the discharge is credible in the presence of such extensive destruction of the tympanum, mastoid antrum, and cells. The erosion of the latter into the sigmoid groove was of old standing, so likewise was the cerebellar abscess. It is probable that the latter may have existed for years, discharging its contents through the middle ear by way of the aperture in the sigmoid groove and the mastoid antrum. The erysipelatous infection had set up fresh inflammatory action and infective basal meningitis which proved fatal. The appearance of the dura over the tegmen tympani was sufficient in itself to account for the serous lepto-meningitis, though it could equally easily have spread from the basal involvement in the cerebellar fossa and about the pons. The case is an illustration of the manner in which pus may discharge from the middle ear and even from the brain or cerebellum for months or years without urgent symptoms, until the discharges become infected by pathogenic organisms, when the most serious disturbance is suddenly lit up.

CASE XLIII. *Case of lepto-meningitis and abscess in the roof of fourth ventricle on middle cerebellar lobe bursting into ventricle. Old standing right otitis media. Death. Coming under observation on third week of acute illness. Duration of acute illness twenty-three days.*

J. C., aged 3 years, was admitted into the Children's Hospital on 13th March, 1889, suffering from cerebral symptoms and facial paralysis in connection with a chronic infective otitis media of right side.

History.—*Heredity.*—Very indefinite history, people poor, child neglected. Patient one of six, all of whom were healthy with one exception—a child who died from "inflammation of bowels." *Personal.*—When a year old patient had an attack of measles followed by whooping cough, during which a purulent discharge occurred from the right ear. About a month later an abscess formed behind the right ear, which was opened, and since that time fœtid pus had continued to flow, both from the opening behind the ear and from the external auditory

meatus. Nine months ago facial paralysis of the right side appeared, at first slight, then gradually developing until it became absolute within three months.

Present Illness.—Notwithstanding this condition the child was able to play about until two weeks previous to admission when he became much worse, lay in bed, refused food, vomited frequently, and seemed very ill. At the same time the discharge from the ear almost ceased.

Condition on Admission.—Patient presented a miserable appearance. He was a weak emaciated child with complete facial paralysis, pale face, pinched features, conjunctivæ congested, especially on the right side. He lay quietly curled up in bed, shrank from observation, wanted to be let alone, resented interference, and, regarding the light an obtrusion, shut the left eyelids; the right remained open as they were beyond his control. Occasionally he gave vent to a sudden sharp screech.

The pupils were variable, often unequal, alternately the right and left became dilated, and when in the dilated state they were stabile or very sluggish. When asleep, as a rule, they contracted. The pupil could be easily observed in the right eyeball as the eyelids remained open during sleep. It was difficult to get the eyeballs to follow the finger, so as to test the efficiency of the ocular muscles. At first this was ascribed to a want of mental concentration, but it was found to be due to a defect in eyesight, which increased subsequently to absolute blindness. Ophthalmoscopic examination showed congested fundi, but no distinct optic neuritis. Otological examination showed that the right membrane had disappeared, a fringe of granulation tissue presenting from the margin of the tympanum. Bare bone was detected beyond this granulation tissue, seemingly in a large cavity in the petrous bone, the tympanum as such having disappeared. An aperture above and slightly behind the mastoid led into this cavity just at the level of the base of the skull, a defect in the latter exposing the dura mater. From both the external auditory meatus and this sinus a discharge of fœtid matter issued. Nurse said she saw a similar discharge issuing from the nose on several occasions. If this fœtid pus came from the ear by way of the Eustachian tube, could it not be swallowed? or, worse still, gain entrance into the trachea and lungs?

When the sinus at the back of the ear was opened it was found that the upper part of the mastoid had disappeared, the base of the skull at the junction of the petrous with the squamous was gone, the dura mater thick and covered with granulation tissue lay bare for an inch and a quarter in length by about three quarters of an inch in breadth. The whole petrous portion was a shell containing bone debris, granulation tissue, and pus. The pulse was markedly intermittent, very weak, and running from about 60 to 80. The temperature was subnormal, the urine very scanty, not albuminous. Respiration irregular and quick. Vomiting frequent.

The patient was closely observed night and day during the next nine days. The marked variability in the pupillary condition continued for the first forty-eight hours. The right pupil would become dilated and almost quite stabile for a period of from two to four hours. It would then gradually give

way, and in turn the left would become widely dilated and fixed. The sight was completely gone, though the media seemed intact; the congestion of the fundi became greater. He could not recognize anyone near him, not even his mother until she spoke. The drinking cup had to be placed on his lips before he recognized what it was. When lifted in bed he was in great distress, crying out "I am falling" and grasping firmly any object within his reach. He could not sit up, and when placed in the nurse's arms in a sitting posture he tended to fall to the right, and would have done so had he been permitted. He could not maintain his equilibrium. Cerebellar ataxia of a marked kind was evident. Though he had not vomited for some time prior to being lifted, yet when lifted he did vomit (which is often the case in cerebellar tumour). On the 15th and 16th his respirations were very irregular and frequent, and on two occasions of a Cheyne-Stokes character. Sighing and yawning were very frequent, the latter being sometimes followed by a sharp screech. The head was retracted, and the muscles of the neck were stiff.

On the 17th and 18th besides the above there was distinct clenching of the teeth, the masseters being very rigid, the retraction of the head being more marked and the muscles of the neck firmer. The power of swallowing was lost. Irregular twitchings of the legs resembling the action of the muscles under electric interrupted current were frequent, the arms were attacked in the same way a few hours later, the muscles of the face were likewise involved, and there were frequent twitchings of the eyelids. In all these parts for many consecutive hours there were constant tremors, and every few minutes there was superadded a series of sharp, quick clonic contractions. The breathing was very laboured, irregular, and many times the rhythm was of the Cheyne-Stokes character. Along with this there was frequent frothing at the mouth. During all this period both pupils were widely dilated and fixed. The temperature within six hours increased from normal to above 102°, and the pulse rose from 98 to 160. The screeching was much more frequent and the perspiration abundant. There was internal strabismus of the left eyeball which lasted for twenty-four hours. During the remaining two days these symptoms remained the same, except that they were modified by the extreme weakness of the patient, who died at 11 p.m. on the 21st March (in 23rd day of illness), having been about nine days under observation.

Appended is the chart of pulse, temperature, and respiration taken every hour for the first day and subsequently every four hours.

Post-Mortem Examination.—On the right temporal bone being examined the mastoid foramen was free ; there was no pus in it or outside of it. The measurement from the external auditory meatus to the tip of the petrous bone was one and three quarter inches; while the depth of the carious cavity within it, measuring from the external auditory meatus, was an inch and a quarter, its greatest breadth being three quarters of an inch. The defect in the base of the skull existing at this part corresponded to the entire length of the petro-squamous suture, and measured three quarters of an inch in breadth and one and a quarter in length. The carious erosions on the floor of

the petrous bone extended into the jugular fossa, and slightly into the carotid canal. The dura mater was in parts thickened and opaque, especially over the petrous and squamous portions of the right temporal, and extending from this along the right side to the middle fossa, ending in the proximity of the optic nerve. There was marked lepto-meningitis, with miliary tubercles scattered freely in the Sylvian fissure, where the lobes and convolutions were soldered apparently by old adhesions. Besides this there was active basal

CHART J.—SUPPURATIVE LEPTO-MENINGITIS.

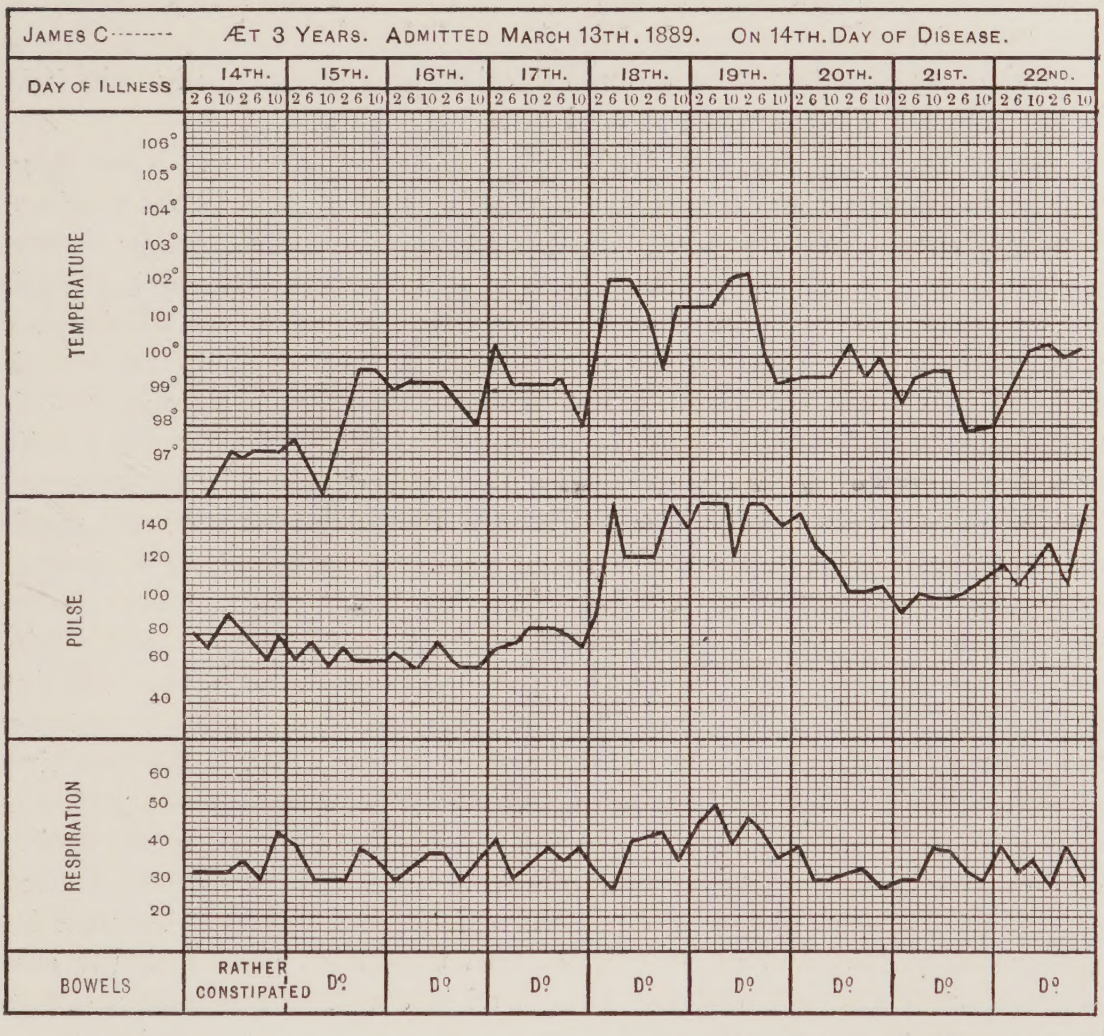

JAMES C·····
ÆT 3 YEARS. ADMITTED MARCH 13TH. 1889. ON 14TH. DAY OF DISEASE.
DAY OF ILLNESS
14TH. 15TH. 16TH. 17TH. 18TH. 19TH. 20TH. 21ST. 22ND.
2 6 10 2 6 10
TEMPERATURE
106° 105° 104° 103° 102° 101° 100° 99° 98° 97°
PULSE
140 120 100 80 60 40
RESPIRATION
60 50 40 30 20
BOWELS
RATHER CONSTIPATED
Dº

Chart of temperature, pulse, and respiration in a case of suppurative lepto-meningitis and abscess in roof of fourth ventricle, bursting into ventricles, arising from right otitis media.—Death.

and cerebellar lepto-meningitis, with considerable soft gelatinous exudation, which was marked at the basal portion of the pons and medulla, surrounding the latter. The cerebral ventricles were greatly distended with fluid, and the fourth ventricle was somewhat occluded by this same gelatinous effusion. On this effusion being removed a superficial abscess was discerned on the roof of the fourth ventricle, involving the middle lobe of the cerebellum, and projecting into the ventricle. The anterior wall of this abscess had disappeared, the posterior alone remaining; it was firm and yellow, being made up of inspissated pus and caseating debris. It was somewhat circular,

and measured over one quarter of an inch in diameter. The fourth ventricle was therefore partly occluded by this abscess, which when intact must have caused absolute occlusion. As it was, the projecting portion of the abscess, coupled with the gelatinous exudation and the congested state of the surface of the fourth ventricle, caused that ventricle to be blocked. Some of the turbid exudation taken from the fourth ventricle showed under the microscope pus corpuscles. Numerous distended bloodvessels were seen on the surface of the lateral ventricles.

The substance of the anterior portion of the brain was pale and œdemic; that of the pons and medulla was congested. The right lateral cerebellar lobe was softened.

Evidences of tubercle were found in other parts of the body, especially in the lungs, where there was one old caseating nodule.

Remarks.—This case presented many interesting features. Apart from the total destruction of the seventh nerve, due to the lesion in the temporal bone, the symptoms were those of lepto-meningitis and cerebellar disease. The meningeal symptoms predominated in the first two days during which he was under observation. On the two following days the blindness,—while the media were intact,—the sensation of falling when set up in bed, the complete loss of equilibration and the falling to the right, coupled with vomiting when he was lifted in the nurse's arms, all pointed to the middle lobe of the cerebellum being involved; the vertigo might also be accounted for in same way or might be attributed to involvement of the semi-circular canals. The succeeding days were marked by symptoms of further cerebellar involvement and possibly of implication of the medulla. These were retraction of the head, rigidity of the muscles of the neck, the respiration rapid and irregular, frequently sighing, and occasionally Cheyne-Stokes in rhythm. Shortly after there was a sudden accession of other symptoms, the sudden rise of the pulse rate and elevation of temperature, the clenching of the teeth, the inability to swallow, the twitching of the limbs with the accompanying shock-like movements, and the stabile mydriasis, all indicating a grave change in the patient's condition, such as could have occurred from the bursting of some abscess and the consequent involvement of the medulla and basal ganglia. It may be granted that this small abscess on the roof of the fourth ventricle did not exist during the time the parents allege he was able to run about, which was a fortnight prior to admission. If it did not, the lesion proved fatal in twenty-three days—five days after it had burst into the fourth ventricle. It is probable that the abscess occurred round the periphery of a tubercular deposition in that spot, but if so there were no symptoms observed indicative of this tubercular nodule in the middle lobe of the cerebellum a fortnight before his admission. It is clear that prior to the formation of the abscess there was old-standing tubercular meningitis, the evidence of which existed in the deposits in the fissure of Sylvius. The havoc wrought in the petrous portion of the temporal bone, the complete obliteration of the auditory apparatus and the aqueduct of Fallopius, leaving but an eroded shell with extensive defects in its walls, through which the dura, the sigmoid sinuses, and the carotid artery

were exposed, must have existed for more than six months. The tegmen tympani had long disappeared, and this large fœtid cavity was in direct contact with the dura, yet there was neither cerebral abscess nor absorption of pus through the sigmoid sinus, setting up infective infarction in the lungs. It is probable that the wall of the sigmoid sinus and dura mater had become chronically thickened and covered by granulation tissue which acted as a barrier to the absorption of infective matter. The stabile mydriasis present during the last few days probably indicated dilatation of the ventricles.

It is possible that the whole disease had arisen primarily from tubercle of petrous bone, spreading thence to the membranes of the brain.

DIAGNOSIS.

Encephalitis and cerebral abscess.—The differentiation between abscess of the brain and acute encephalitis is impossible at an early stage, both being present coincidently, the abscess developing from the encephalitis. An encephalitis which has originated from an infective source will probably end in suppuration, and especially so if the encephalitis lasts over a fortnight. But the time element is not to be relied on in distinguishing between encephalitis and abscess unless coupled with the above ætiological factor. Encephalitis developing from a mechanical cause may exist for a much longer period without pus production, and when suppuration does occur it must be from superadded infection. Were a rigor supervening, the probability of abscess formation would be increased. A comparatively small source of infection, especially when situated in the neighbourhood of the skull, is sufficient to occasion infective embolism ending in abscess.

Diffuse lepto-meningitis and cerebral abscess.—The differentiation between cerebral abscess, suppurative lepto-meningitis, and disintegrating thrombosis of the sinuses presents difficulties, especially in complicated cases. They may all arise from the same source, and any two, or all three, may be present in the same case at the same time. There is generally a degree of lepto-meningitis, more or less limited, accompanying cerebral abscess, and some of the symptoms attributed to abscess may in reality be due to the accompanying meningitis. Diffuse lepto-meningitis supervenes toward the terminal period of abscess when the latter has burst upon the surface of the brain. In this event the symptoms of lepto-meningitis prevail, and unless the history of the case is distinct, the indications of the abscess may then be masked.

When diffuse lepto-meningitis is present the high temperature, without the marked remissions characteristic of infective sinus thrombosis, the rapid pulse, the general irritability and over-acuteness of all the senses are in marked contrast to the symptoms of uncomplicated cerebral abscess (Temperature, Pulse, and Respiration Charts, see chapter on "Symptoms" under heading Temperature). When marked encephalitis is present along with a degree of lepto-meningitis the pulse is at least slower than in traumatic or infective lepto-meningitis alone, though the temperature is above normal. This is illustrated in Case XXVII., where there were symptoms of basal lepto-meningitis implicating the cerebellar fossa and upper part of the cord. On recovery from the lepto-meningitis, the symptoms of encephalitis were for the first time unmasked, and subsequently abscess developed. Lepto-meningitis implicates the cranial nerves in a much more general and erratic manner than cerebral abscess. Optic neuritis is also present in some cases of lepto-meningitis. When the meningitis is distinctly limited to the area over the abscess, then the symptoms of the abscess predominate.

Infective thrombosis of the sigmoid sinus and abscess.—The symptoms of infective thrombosis of the sigmoid sinus, especially when the thrombus is disintegrating, are distinctive of the affection. The high temperature, with its marked remissions, rapid weak pulse, repeated and increasingly recurrent rigors, followed by profuse perspirations, the symptoms of pulmonary infarctions, colliquative diarrhœa, pain in the upper third of the posterior triangle, submastoid region, and down the course of the affected jugular, are often marked. The other variety of symptoms accompanying certain forms of sinus thrombosis is rather of a typhoid type, and such cases have not infrequently been diagnosed as enteric fever, and therefore the symptoms would be unlikely to be confounded with those of cerebral abscess, though they may mask the abscess when it is present.

When cerebral abscess is associated with this condition, even when all three—lepto-meningitis, abscess, and infective thrombosis—are present in the same case, the symptoms of the infective disintegrating thrombosis are so pronounced as in measure to obscure the presence of the others. The basal suppurative lepto-meningitis still manifests itself in the retraction of the head and neck and in the affection of the intracranial nerves. In many such instances the cerebral abscess is small, and secondary in

point of time to the thrombosis, though originating from the same source. Occasionally this order is reversed. When infective thrombosis is present along with abscess, it is by far the more serious of the two, and demands the earlier attention. The recognition of the abscess, were it possible in such a case, and its evacuation, would not of themselves be likely to cure the patient.

When the system has become poisoned by infection the pulse is weak and very rapid, and a point in the diagnosis of this condition is found in the fact that in such persons the administration of narcotics such as chloroform or opium in no way lessens the rapidity of the pulse. When complete anæsthesia is established, the pulse continues at the same rate as before the administration was begun; whereas in rapid pulse from excitement the narcosis causes marked diminution in the rate.

Tumour and abscess.—It is sometimes necessary to discriminate from the symptoms between abscess and tumour of the brain. The absence of any pathogenic source from which the encephalitis might arise as a sequela, the slow progress of the symptoms, the more definite focal phenomena, the slow and progressive involvement of the cranial nerves, the intense degree of optic neuritis, the prolonged periods during which the symptoms greatly lessen, according to the variation in the intensity of peripheral encephalitis, the absence of rigors, all point to tumour. The temperature and pulse are of little value in discriminating between these two affections, except that subnormal temperature points rather to abscess.

Prognosis.—Cerebral abscess must always be regarded as a serious affection imminently dangerous to life. The great majority of cases not dealt with surgically end in death within a short period, generally a few weeks. On the other hand, there is no cerebral affection more amenable to surgical treatment, and none which offers better results. An uncomplicated cerebral abscess, whose position is clearly localized, if surgical measures are adopted for its relief at a sufficiently early period, is one of the most hopeful of all cerebral affections. It is much more so than suppurative lepto-meningitis, or even than infective thrombosis of the sigmoid sinus, unless the latter can be operated on before the disintegration and dissemination of the infective contents of the vein take place. After aseptic evacuation of the abscess, not only is the patient likely to recover, but in many instances it leaves no perceptible permanent mental or bodily damage. Within

a few days, sometimes hours after the evacuation, the mind becomes bright and intelligent, the paralysis passes away, and within a few months the optic neuritis has disappeared, leaving the visual function unimpaired.

Occasionally, after evacuation of the abscess, there may be some soldering of the brain to the skull, or to the almost equally rigid dura mater or fibrous covering filling up the trephine aperture, though this is much less than what occurs after the removal of tumours. When this occurs the patient may be subject to occasional faintness on sudden exposure to great heat or on sudden exertion. One man, a blacksmith to trade, says that after he had been cured of his abscess he occasionally felt on sudden effort, especially when beside the furnace, a faintness coming over him, which caused him to drop what he held in his hands and to lie down for a moment. This occurred several times the first year, and seldom afterwards, as he carefully avoided exposure to great heat or *sudden* exertion. He was capable of strenuous and prolonged exertion provided it was not sudden.

The prognosis without operation will in some measure depend upon the size of the abscess, the smaller ones occasionally being absorbed, the larger ones usually ending in death. The position of the abscess in the brain will also affect the prognosis. If it be situated deeply, so as to involve the more vital parts, death will speedily follow. On the other hand, a superficial abscess not operated on may be evacuated, if a sufficient vent is formed for it by pathological processes through the dura and bone. This, however, is rare, and even those so connected with the exterior of the skull generally end fatally sooner or later.

Even an abscess which has become encysted, and has remained for a considerable period without evincing any signs of its presence, may be a source of danger. The encysted abscess may burst, or it may act as a foreign body round which fresh inflammation is set up (see Case XXX., "Abscess of Brain"), and if situated in the cerebellum may cause death by œdema of the brain and serous distension of the ventricles. An encysted abscess may, however, become absorbed; but until it does so it is a menace to the patient's life, as it is still apt to have fresh suppuration induced in its vicinity whenever fresh organisms are introduced, probably gaining access by the original channel of infection. The source from which the abscess sprang remaining active is an additional element of danger, both by way of occasioning fresh suppuration

round the abscess, and also by creating fresh foci of inflammatory disturbance, such as sinus thrombosis or lepto-meningitis.

The cure of cerebral abscess depends on its early detection, accurate localization, and speedy and efficient evacuation under aseptic precautions.

Course of cerebral abscess.—The time occupied by cerebral abscess in developing and pursuing its course when uninterrupted by treatment is variable. Acute abscess runs its course in from two to six weeks, whereas chronic cerebral abscess, when encapsulation takes place, may last for a very long period—months to many years. As a rule, abscess arising from infective otitis media runs a more rapid course than abscess arising from infective traumatism, without direct exposure of the brain. No doubt the nature of the specific organism introduced influences the duration of the course.

CHAPTER IV.

THROMBOSIS OF THE INTRACRANIAL SINUSES.

Introductory note to Chapter.—Abercrombie was among the first to draw attention to thrombosis of the intracranial sinuses. In his volume on *Diseases of the Brain*, case 5, he relates the symptoms and pathological appearances of an instance of thrombosis of the lateral sinus observed by him in 1816. Many papers have since been published on this subject. Bouchut in 1845 drew attention to the influence of marasmus in determining coagulation of the blood of the veins of the inferior extremity, and pointed out the probability of thrombosis sometimes occurring in the sinuses of the dura mater during convalescence from enteric fever. Since then numerous observations of marasmic thrombosis in the longitudinal sinus have been published. Von Dusch gives an excellent paper which was translated into English in 1861. In the ophthalmological journals there are numerous cases related of thrombosis of the cavernous sinus arising principally from inflammation originating in the ophthalmic veins. Knapp,[1] Lapersonne,[2] Coupland,[3] have contributed valuable information concerning cavernous sinus thrombosis; Verneuil, Reverdin, and others, have furnished instances of anthrax of the face and lips giving rise to cavernous sinus thrombosis through the medium of the facial and ophthalmic veins. Thibault contributes an instance in which the cavernous sinus was implicated through the ophthalmic vein from a nasal lesion. A case of periosteal 'phlegmon' of the jaw arising from dental caries causing suppurative phlebitis

[1] Knapp was the first to clearly recognize thrombosis of the cavernous sinus, giving three cases in the *Archiv. fur Ophthalmologie*, 1868, t. xiv. p. 220.

[2] Lapersonne, *Archiv. Ophthalmologie*, 1885.

[3] Coupland, *Ophthalmological Society's Transactions*, 1887.

of the sinus was communicated by Demons of Bordeaux, and Panas gives an instance where thrombosis of the intracranial sinuses was brought about from gangrene of the tonsil. There is also an excellent monograph in French, compiled by Lancial,[1] who has collected a large number of cases from which much assistance has been obtained.

SINUS THROMBOSIS.—Venous thrombosis is especially met with in one of three localities, which, in the order of their relative frequency of involvement, are, intracranially, in the true pelvis, and in the lower extremities.

The construction of the sinuses predisposes them to thrombosis. Their great width, the rigidity of their walls, their somewhat triangular form, the trabeculæ which occasionally cross them, the peculiar manner in which they are prevented from being emptied during inspiration, and in the case of the longitudinal sinus, the direction in which the blood from the cerebral veins enters at an obtuse or right angle against the current, all tend to retard the flow of the blood and thus to favour coagulation. When there is added to these conditions a deficient supply of blood, occasioned by exhaustion or depletion, such as from profuse diarrhœa, then marasmic thrombosis is apt to form.

Two forms of thrombosis.—There are two recognized forms of thrombosis, primary or marasmic, and secondary or infective. The latter is sometimes called inflammatory. Although the marasmic variety of thrombosis is not an infective disease it has been included here to contrast with the infective variety and to make the view of intracranial thrombosis more complete.

MARASMIC THROMBOSIS occurs much less frequently than the inflammatory, and is almost invariably located in the longitudinal, rarely in the lateral, and still more rarely in the cavernous sinus. The marasmic form occurs in weakly people, or in those who have been prostrated by exhausting diseases. It is most often observed at the two extremes of life. In the young, though it may occur till the age of fourteen years, it is met with most frequently during the first two years, and more especially during the first six months of

[1] Louis Lancial—(Paris, G. Steinheil, 1888)—*De la Thrombose des Sinus de al Dure-Mère.*

infancy. Exhausting diarrhœa is one of the most potent causes in children, though acute and chronic pulmonary affections also play an important ætiological part. In numerous reported cases of marasmic thrombosis occurring in childhood, respiratory obstruction, with consequent dilatation of the right side of the heart, was the primary cause. Among these were lobar and lobular pneumonias, pleuritic effusion, and exudation with ultimate thoracic contraction, and tuberculosis of the lungs. Though marasmic thrombosis may occur during the progress of such diseases, it usually does not occur till toward the end of the attack or until early convalescence. In adults, exhaustion following enteric fever, phthisis, and the puerperal state, are frequent causes, and in old people it occasionally follows carcinomatous marasmus.[1]

In all of these states, the circulation is weakened by the diminished force of the heart, the blood becomes inspissated, and it exhibits a greater tendency to coagulate. Marasmic thrombosis is prone to occur in the longitudinal sinus, where the circulation is very slow, and the chief cerebral veins, which of themselves have a retarded flow, pour their blood into the sinus against its stream. Thrombosis may also arise in one of the cerebral veins, to which it may be limited, or it may spread thence into the longitudinal sinus. When thrombosis involves, or is limited to, a cerebral vein, it may give rise to symptoms indicative of localized cortical lesion, such as unilateral convulsions, followed by a monoplegia or hemiplegia. In some such cases, post-mortem examinations have shown thrombosed veins in the motor cortex. In infants, profuse diarrhœa diminishes the quantity of blood, and removes a large portion of serum from the brain, as well as from other parts of the body, consequently there is a diminished volume of the cranial contents, shown at first by depression of the fontanelles, and subsequent overlapping of the cranial bones at their sutures. In this state the cerebral vessels and sinuses are apt to dilate, causing a further retardation of the already slow flowing stream, which, coupled with the enfeebled heart's action, and the inspissated blood, tends to establish thrombosis of the sinuses.

Pathology.—Marasmic thrombosis, though generally confined to the longitudinal sinus, may extend into other sinuses, so that the lateral, sigmoid, and even the cavernous sinuses and the jugular

[1] In a specimen in the possession of the author, the longitudinal sinus, and one lateral sinus, were found thrombosed, the patient dying from carcinomatous marasmus.

veins, may become implicated in the extension. In a few only of the fatal cases examined was the superior longitudinal sinus alone found to be affected with marasmic thrombosis. It must not be inferred from this that marasmic thrombosis is not frequently limited to the longitudinal sinus, but only, when so limited, death does not so often occur as when it extends into the lateral sinuses. In the majority of cases reported, when it did extend from the longitudinal, both lateral sinuses became affected. In one case seen by author at post-mortem, the superior longitudinal and *one* lateral—the larger of the two—were affected with marasmic thrombosis.

In marasmic thrombosis the clots are dense, resistant, stratified, and non-adherent to the walls of the vein.[1] They seldom occupy the whole lumen of the vessel; they tend to become organized or absorbed, and very rarely disintegrate. In chronic cases the clot is found tunnelled sufficiently to permit the re-establishment of the circulation. As a consequence of the thrombosis, great congestion of the meningeal and cerebral vessels ensues, accompanied by œdema. The amount of congestion and œdema depends upon the extent and situation of the clot. If it be confined to one sinus, a portion of the blood can still pass by some of the larger tributary veins, thereby lessening the congestion and œdema, but if the clot has extended into one of the cerebral veins, the return flow of blood from that portion of the brain is greatly impeded. Hence, not only is the congestion and œdema great, but the capillaries in the affected area of the cerebral cortex burst, giving rise to innumerable minute hæmorrhages, which, along with the congestion and œdema, occasion cerebral softening of a red colour. In severe cases the ventricles become distended with serous fluid, and, exceptionally, the sero-sanguineous effusion passes into the retro-ocular tissue, causing exophthalmos. As a rule the frontal lobes are least affected by this softening, the parietal and occipital most. Meningeal hæmorrhages from marasmic thrombosis do occur, though they are uncommon. If the patient recovers, atrophy and induration of the affected area is believed to result.

Symptoms of marasmic thrombosis.—The symptoms of marasmic thrombosis are often uncertain, and are prone to be masked by the diseased condition which precedes and is the cause of the thrombus. The symptoms to which it gives rise are those of

[1] A post-mortem clot is black or yellowish-white, and very friable, easily breaking down under the fingers.

venous obstruction, intra- and extra-cranial, and perverted function of the affected part of the brain. As the longitudinal is the sinus most often affected in marasmic thrombosis, venous distension of the frontal and parietal veins accompanied by œdema is the most frequent external sign. Occasionally epistaxis occurs from over-distension of the veins communicating with the anterior part of the longitudinal sinus, when they are not involved in the thrombus. Strabismus, tremors of the lower limbs, contracture and rigidity of the muscles of the extremities, and occasionally of those of the neck and back are frequent, and are probably due to meningitis. Children affected with marasmic thrombosis are prone to convulsions, which seldom occur in adults, severe headache and delirium being in the latter the expression of the same condition. The convulsions are usually general, and are accompanied by unconsciousness. As convulsions are so common in children, originating from such a variety of causes, Bouchut observes that convulsions occurring at the beginning of an acute malady are not of serious import, usually heralding one of the exanthemata or a phlegmon; but when they occur at the end of an acute and exhausting disease, or during a chronic illness, which has greatly reduced the little patient, then marasmic thrombosis is to be feared. Occasionally the convulsions are unilateral, and may even be confined to one member. In such a case it is probable that the cerebral veins, from the motor cortex, have become thrombosed as well as the sinus. The convulsions are paroxysmal and intermittent in character; the usual duration of the series is from a few hours to two days, but they may occur at intervals during several weeks.

Thrombosis of the retinal vessels occasionally occurs.

The diagnosis of marasmic thrombosis in the adult is difficult, and is seldom definitely determined during life. If in adults of an age when cerebral lesions are not to be expected, an exhausting illness is followed by a sudden cerebral lesion, especially if unilateral, and accompanied by headache, delirium, somnolence, and coma, there is a probability of marasmic thrombosis, which is increased by the presence of œdema and distension of the veins of the parietal and frontal regions of the scalp. In children, convulsions occurring towards the end of exhausting illnesses, œdema, and venous congestion of the parietal and frontal portions of the scalp, epistaxis, and symptoms of a localized brain lesion, render a diagnosis of marasmic thrombosis probable. In infants,

these symptoms are often accompanied by depression of the fontanelles and by overlapping of the cranial bones.

INFECTIVE THROMBOSIS.—*Inflammatory thrombosis* is the term usually applied to thrombosis which arises from invasion of the sinuses by pathogenic micro-organisms; therefore it would be more correctly described as infective thrombosis.

Infective or *inflammatory thrombosis* occurs chiefly in adults, rarely in old people, and only exceptionally in children. It generally affects one of the dual sinuses, while marasmic thrombosis affects the single median or azygos sinuses. It is local in origin, secondary to some inflammatory lesion of infective character, and occurs in the sinus nearest the seat of the primary disease. Infective thrombosis is often associated in its later stages with meningitis, and not infrequently with small cerebral or cerebellar abscess.

Infective thrombosis may arise from traumatic lesions which induce infective inflammation such as occurs in the diploë of the skull from dirt-ingrained, punctured or comminuted fractures, or even from infective scalp wounds without fracture. The most frequent cause, however, is middle ear disease and the destructive processes arising therefrom, therefore the sigmoid sinus is the one most commonly affected with infective thrombosis, but pathological lesions, situated anywhere about the head and face, may give rise to thrombosis. Anthrax, when it affects the lips, mouth, nasal fossa or orbital cavity, is a common cause. Erysipelas anywhere about the face and head, infective cellulitis in the orbital cavity, infective ulceration in the nasal fossa and pituitary mucous membrane, periostitis of the jaws arising from dental caries, inflammation of the tonsils and retro-pharyngeal abscesses, have all been noted as causing thrombosis, usually of the cavernous sinus.

Infective thrombosis may originate from local disease in one of two ways. The inflammatory action may extend by contiguity of tissue from the seat of disease to the sinus, whose walls become inflamed, the blood in its interior coagulates and adheres to the inner coat, and thus phlebitic thrombosis is established. Secondly, a small vein at the seat of disease may become affected by thrombosis, which extends along this vein into the sinus, causing obliteration of the latter. In both of these the cause of the inflammation is the propagation of infective elements along the lumen of the vein or through the neighbouring tissues.

There is a third cause alleged, namely, pressure on a sinus, such as might arise from a tumour. This must be a rare cause of thrombosis, and cannot produce inflammatory thrombosis, as pressure pure and simple by a tumour produces obliteration of a vein without inflammatory action.

Pathology.—When infective thrombosis affects a sinus, its endothelium becomes desquamated, its walls softened, thickened, and sometimes so much disintegrated that an hiatus forms in them. Hæmorrhage would then ensue, were it not for the fact that coagulation of the blood occurs within the vessel coincident with the extension of the inflammation to its walls, or at least with the endothelial desquamation. Hence in advance of the disintegration or ulceration of the walls of the sinus, the lumen of the vessel is occluded with blood clot, so that hæmorrhage rarely occurs in infective thrombosis. The inflammation of the wall of a sinus causes a blood clot to form and to adhere closely to the coats of the vessel. As the inflammatory thrombosis is as a rule due to infective processes, the clot soon disintegrates, pus formed from the vessel wall mingles with it, and produces a greenish brown, grumous particulate fluid. This fluid swarms with living organisms which, having found abundant pabulum for their proliferation in the blood clot, are apt to be carried into the circulation. This may be effected in several ways. First, minute infective particles may pass into the descending blood stream through the tributary veins in the vicinity of that part of the sinus which is filled with disintegrating clot, the reversal of the current in these tributary vessels owing to the occlusion of the sinus, permitting the dissemination. Secondly, if the disintegration of the thrombus is so complete as to permit the partial re-establishment of the stream through the thrombus, then the softened particles may be swept into the general circulation. In some post-mortem examinations of infective sigmoid sinus thrombosis, though a firm clot was found occupying the position of the sinus nearest the arteries, the remainder of the clot had evidently undergone extensive softening and disintegration, and had been partially removed, as detached ragged particles of it were found adhering to the walls of the vessels for several inches nearer the right side of the heart than the thrombus. The sinus was therefore practically patent on the side nearest the internal jugular. Thirdly, infective matter may spread through the walls of the sinus into the neighbouring parts, where it may be taken up

by the congested vessels and perivascular spaces and carried into the circulation.

The internal jugular, especially its upper third, not infrequently —in the later stages of the affection—participates in the inflammatory action, and the same series of phenomena may be seen within it as occur in the sinuses from phlebitis—thickening of the coats of the vessel, desquamation of its endothelium, and adhesion of minute portions of clot, to the formation of a complete thrombus, with sequent disintegration and abscess formation. Many of the large venous channels leading into and from the sinus, such as the superior petrosal sinus and the posterior and anterior condylar veins, participate in the thrombosis, and may convey disintegrated infective clot into the circulation.

As a result of the infective thrombosis within the vessels, the tissues in their periphery inflame, soften, and are filled with exudation, which owing to infective influences becomes disintegrated. Intracranially, the phenomena differ somewhat from those which occur outside the skull; probably owing to the greater thickness and power of resistance of the walls of the sinus as compared with those of the veins, and from the sinus being partly embedded in bone. Where the walls of the sinus are most affected, peripheral exudation occurs, generally in a plastic form, which, were it to become organized, would be of advantage to the damaged vessel, as the weakened wall would be strengthened by a fresh peripheral formation of connective tissue. Again, when it occurs on the cerebral side of the sinus, were the exudation organized, firm adhesion between the soft membranes and the dura would result, and would be a safeguard against diffuse lepto-meningitis. Neither of these desirable results as a rule occurs, as the exudation is subjected to the same disintegration as the thrombus within, and instead of this effusion aiding the diseased vessel it weakens it, and is a source for the dissemination of the infective matter. On the side of the skull the exudation forms into a purulent layer, stripping the dura from the bone. The macerated wall of the sinus, deprived of its blood supply by the pus within and without, sloughs, and then its purulent contents mingle with the pus on the exterior of the sinus. When the processes described occur in the sigmoid groove, the pus may find an exit by the mastoid foramen outside of the vein which passes through it, and form an extracranial abscess at the posterior aspect of the mastoid. Occasionally the disintegrating thrombus may have extended into this vein,

and an intravenous purulent focus forms in the neck on the outside of the skull. The same infective processes may extend through the posterior condylar foramen intra- or extra-venous, and form an abscess which is generally localized under the deep fascia in the upper third of the posterior cervicle triangle. When the pus has formed on the visceral side of the sigmoid sinus, it may find an exit through the anterior condylar foramen by the side of the perineurium of the twelfth nerve, or along the course of the accompanying vein which communicates with the vertebral venous plexus. When, however, the visceral layer of the sinus becomes affected acute lepto-meningitis usually forms, though, when the infective action is mild, the meningitis may be localized and a layer of yellow pus form in the meshes of the arachnoid covering, and probably partially solder it to the sigmoid sinus. Should the infective thrombus extend into the internal jugular, abscess may form in the neck in the vicinity of the jugular. When the cavernous sinus is the seat of the disease, the orbital cellular tissue especially along the course of the ophthalmic vein is usually the site of purulent collections.

Effects of infective thrombus on the neighbouring bone and soft tissues.—It has been seen that the infective inflammation seated in the bone may extend from the initial focus into the sinus, causing thrombosis therein. On the other hand, the infective thrombus, during its disintegrating period, produces a marked effect upon the bone in contact with it. This, in many instances, is produced without the formation of pus outside the sinus. The bone at these parts becomes deeply pigmented, of a dark greenish or brownish hue, and its surface is roughened or eroded. The staining is probably due to the action of the bacillus pyocyaneus or other chromogenic organisms. By these markings on the bones of the skull and vertebræ the position of the thrombosed sinus and veins may be traced, and so deep is the staining that it persists after the soft parts have been removed and the bone subjected to prolonged immersion in water. The erosion, when it occurs, is indelible. The soft tissues in the vicinity also participate in the staining. The lateral and sigmoid sinuses, when the seat of infective thrombosis, leave a stain on the cerebellum, which accurately marks their relative positions. The course of the internal jugular, posterior condylar, and vertebral veins, is mapped out in a similar manner upon the bones and soft tissues on the exterior of the skull. When the patients live sufficiently

FIG. 52.—POSITION OF SIGMOID SINUS MARKED BY PATHOLOGICAL PROCESSES ON CEREBELLUM.

Dark staining, marking part of cerebellum upon which the sigmoid sinus rested. Base of brain taken from case of infective sigmoid sinus thrombosis. 1. Mark of sigmoid sinus.

To face page 234.

long, softening of the surrounding tissues ensues at the stained parts. This was well seen in one case where softening of the dark stained cerebellar tissue adjacent to the affected sinus had occurred.

Besides the local effects of infective thrombosis, there is the much more serious one of systemic infection. During the disintegration of the thrombus, particles of it are carried into the general circulation, and arrested principally in the minute vessels of the lung. Infarctions form, frequently ending in pulmonary abscess, complete pulmonary disintegration, or in gangrene of the lung, often preceded by infective pneumonia. The pleura and pericardium frequently become secondarily implicated.

After the sinus has been thrombosed, if the infective process has been so mild as to cause very limited disintegration, a formative process may be established in the wall of the vessel. Granulation tissue sprouts from the wall of the sinus and the adjacent dura mater and penetrates into the osseous erosion on the one side, and into the lumen of the vessel on the other. If time be allowed the sinus will become obliterated by fibrous tissue at this point. As long as the granulation tissue remains intact it acts as a barrier against the entrance into the sinus of the infective pus which occupies the erosions in the mastoid process, and which would otherwise excite disintegration in the vessel wall. If anything occurs to detach the granulation tissue, and to expose a raw surface in the midst of infective pus, the latter may gain access into the vascular system, through the open mouths of the abrupted bloodvessels. Not only do cerebral and cerebellar lepto-meningites arise as a sequel of such abruptions, but thrombosis of a disintegrating kind with pulmonary infarctions has been known to follow the removal from the sigmoid sinus of such protective granulations.

A thrombus originating in infective processes does not necessarily become totally disintegrated. Probably the inflammatory action set up in the wall of the sinus produces such an extensive thrombus that a portion of it is placed, temporarily at least, beyond the limits of the infected area. This may be detected in the internal jugular, when a cord-like mass may be felt under the sterno-mastoid, to the outer side of the carotid, extending for some inches nearer the right side of the heart than the softened disintegrated thrombus which distends the vessel nearer the sigmoid sinus. The latter may even form an abscess which may burst externally, and yet the organized thrombus below that point may have occluded the vein so perfectly as to prevent the lungs and

other organs from being invaded through that channel. In several instances, the anterior wall of the sigmoid sinus has been destroyed, fluid pus and disintegrated blood clot lying between the internal wall of the sigmoid sinus and the sigmoid groove, yet the internal jugular has been found to be hard from thrombic occlusion, and the lungs found not affected, operation upon the sigmoid having prevented the further spread of mischief.

It is recorded that not only may the internal jugular be the seat of large disintegrating thrombic abscess, but the superior vena cava may likewise be involved.

Cerebral and cerebellar abscesses are apt to form by extension of pathogenic processes from the visceral side of the sinuses. This is most frequently seen in the cerebellum from the sigmoid. From the same cause basilar purulent meningitis is apt to arise.

Though the liver and kidneys may be the seat of infective infarction, it is the lungs which principally intercept the infective emboli. In the majority of instances the liver and kidneys are free even when the lungs are totally disintegrated.

The salient points of difference between marasmic and infective thrombosis may thus be stated :—

Marasmic.	Infective.
1. Chiefly affects the azygos sinuses.	1. Chiefly affects the dual sinuses.
2. The clots tend to organization or are absorbed.	2. Clots tend to purulent disintegration.
3. Hæmorrhages into cerebral cortex in about half the cases.	3. Hæmorrhages into brain or cerebellum seldom occur.
4. Tendency to produce brain softening.	4. No tendency to brain softening.
5. There is seldom purulent infection as a sequence.	5. Purulent infection common, septic or infective emboli.
6. No accompanying lepto-meningitis, cerebral or cerebellar abscess.	6. Often co-incident purulent lepto-meningitis, cerebral or cerebellar abscess.

Symptoms.—Infective thrombosis does not give rise to any one pathognomonic symptom. Locally there is circulatory disturbance, causing interference with the cerebral function and extracranial œdema. The general symptoms are such as may arise from the systemic effects produced by the dissemination of infective organisms or disintegrated blood clot and pus.

General symptoms.—There are certain symptoms which are common to all cases of infective thrombosis. Cephalalgia is one

of the earliest symptoms, and though it may be seated at any part of the head, is often referred by the patient to the region of the primary disease and to the affected sinus or its neighbourhood. The pain is frequently so severe as to prevent the patient from obtaining rest during the first twenty-four or forty-eight hours. Vomiting is a frequent accompaniment. The temperature rises rapidly, sometimes reaching 103° to 106° F., but is subject

CHART K.—INFECTIVE SINUS THROMBOSIS.

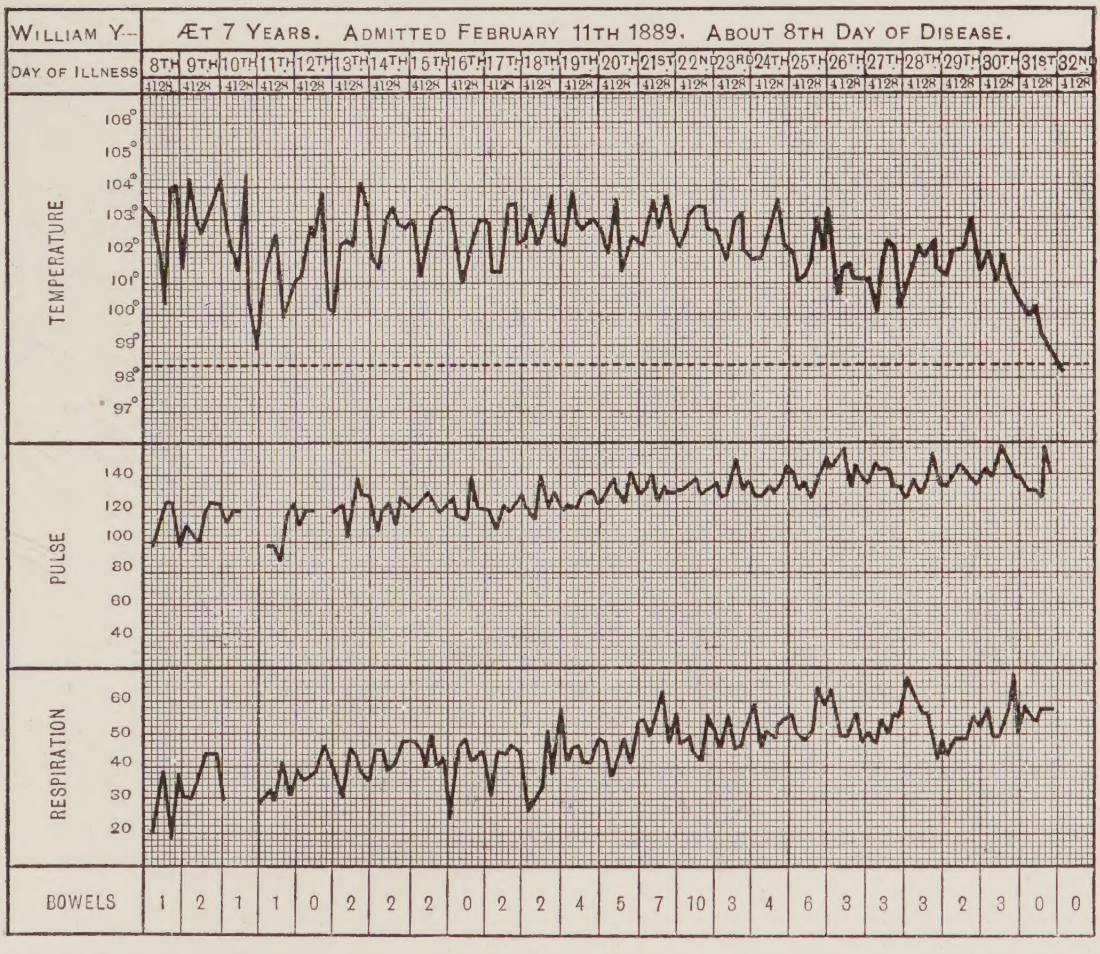

WILLIAM Y.—
ÆT 7 YEARS. ADMITTED FEBRUARY 11TH 1889. ABOUT 8TH DAY OF DISEASE.
DAY OF ILLNESS
8TH 9TH 10TH 11TH 12TH 13TH 14TH 15TH 16TH 17TH 18TH 19TH 20TH 21ST 22ND 23RD 24TH 25TH 26TH 27TH 28TH 29TH 30TH 31ST 32ND
4 P.M. 8 P.M.
TEMPERATURE
106° 105° 104° 103° 102° 101° 100° 99° 98° 97°
PULSE
140 120 100 80 60 40
RESPIRATION
60 50 40 30 20
BOWELS
1 2 1 1 0 2 2 2 0 2 2 4 5 7 10 3 4 6 3 3 3 2 3 0 0

Chart of temperature, pulse, respirations, in a case of purulent lepto-meningitis arising from infective otitis media (left).

to marked depressions. It thus fluctuates, and is often of a markedly remittent type. The pulse is rapid, small and thready, and retains this character—as in all infective diseases—when the patient is deeply narcotized; thus, in a patient placed deeply under the influence of chloroform, the pulse retains its rapidity. Rigors are a prominent feature; they occur early, are often repeated, in some forms, every second or third day throughout the attack, and they exhibit a tendency to increase in frequency

toward the termination of the illness. They are followed by profuse perspiration, such as one sees in pyæmia. The tongue remains dry and coated, and the mouth emits a heavy fœtid odour. There is complete anorexia. The bowels may be constipated, though diarrhœa is the rule, especially toward the terminal period. Besides these symptoms, common to all cases of infective thrombosis, there are others, which may be arranged into three groups,

CHART L.—INFECTIVE SINUS THROMBOSIS.

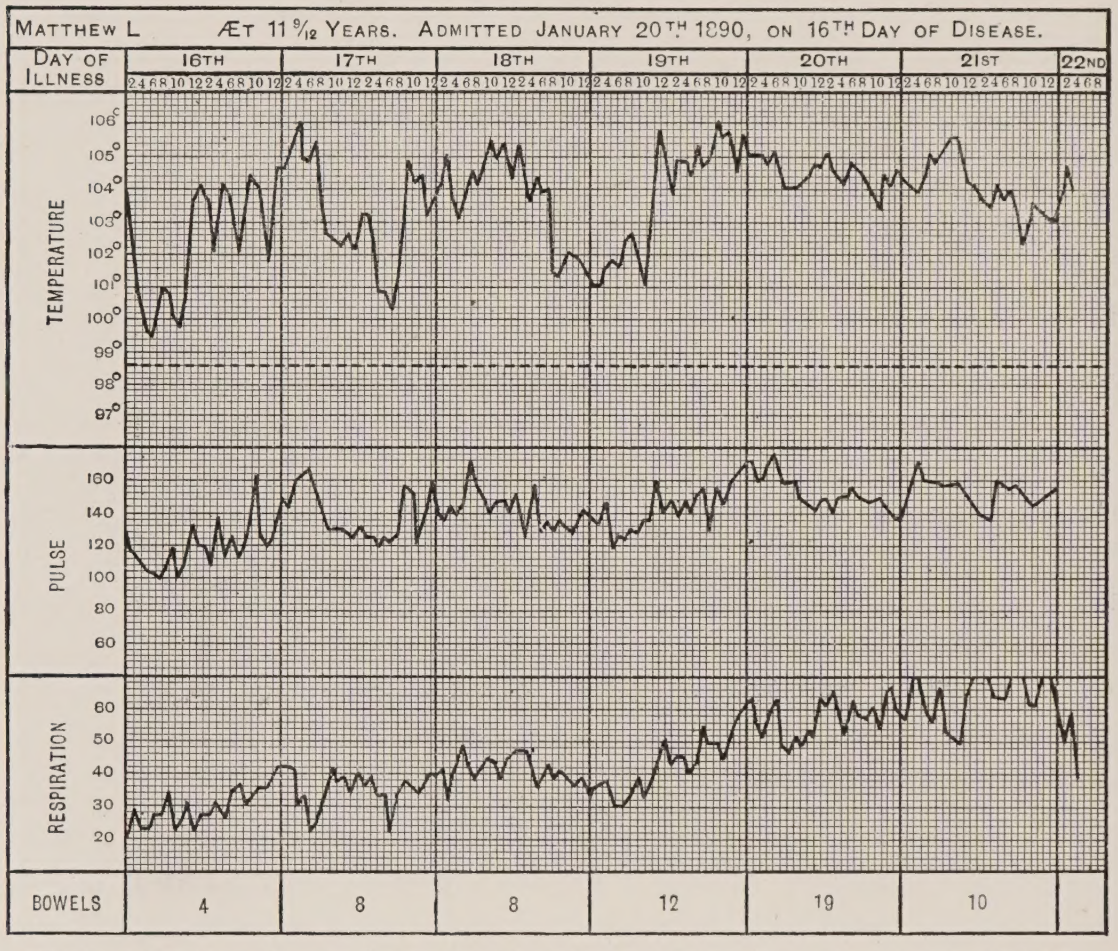


Chart of infective thrombosis of sigmoid sinus—abscess in the temporo-sphenoidal lobe—systemic infection—arising from left otitis media. Death.

according as the dominant symptoms are pulmonary, abdominal, or meningeal.

Pulmonary type.—After a variable interval, but frequently toward the end of the second week, the lungs become involved very insidiously, the other symptoms masking the implication. In many, the first symptom of pulmonary mischief is slight dyspnœa. The patients frequently have a cough though they are unaware of it. Sometimes they complain of a localized 'stitch' in the chest occurring suddenly, often very severe

and lasting several hours, after which the pain at that particular spot abates. In a short time pain of a similar character occurs at another part, either in the same or in the opposite lung, which follows the same course and then subsides. As the case proceeds there may be two or three spots in the lung complained of at the same time. Occasionally the patient believes he can cover the painful spot with his finger tip, but often the pain is described as more diffuse. These sudden painful sensations are probably due to plugging of the minute pulmonary vessels and the formation of infarctions scattered over the lungs. In the first twenty-four hours, during which these 'stitches' occur, physical examination of the lungs made by acute diagnosticians may give negative results. But twenty-four hours subsequently, and occasionally much sooner, "prune juice" expectoration occurs, and then, or shortly after, coarse râles are found here and there, to be followed in another twenty-four hours by moist râles all over the greater part of one or both lungs.

The expectoration at the outset is "prune juice" in colour, but it soon assumes a brownish-grey shade from purulent admixture. It is found to swarm with organisms, and has a putrid odour which increases in offensiveness as the case proceeds.

Occasionally the amount of purulent secretion is very large, and so offensive that the patient has to be isolated and extensive fumigation employed for the comfort of the attendants. The same fœtor is emitted from the exhaled air, and should the patient have diarrhœa—which is frequent as the case advances—the same odour is given off from the stools.

The lungs may be the seat of infective pneumonia, passing on to gangrene, or they may contain numerous infarctions produced by infective emboli, which cause abscess and widespread disintegration. The pulmonary symptoms will therefore vary according to the special form which the inflammation in the lungs assumes. Acute pain in the hepatic region is spoken of as a symptom. It occasionally does arise, probably from congestion of the liver, but also possibly from extension of pulmonary inflammation through the diaphragm. Hepatic abscess rarely occurs from this cause.

In marked contrast to cases of uncomplicated cerebral abscess in which there is slow cerebration, hazy comprehension, and marked want of sustained attention, the cerebral function in uncomplicated cases of infective thrombosis is active and unim-

paired. The intelligence is acute nearly to the end. The duration of the disease is variable, from two to four weeks. Death most frequently results from exhaustion, though coma may ensue.

Typhoid or abdominal type.—The toxæmia sometimes manifests itself in the abdomen, when a marked typhoid condition is developed. The initial symptoms are much the same as those just related above, with the exception that rigors, though they occur, are fewer, and by no means a prominent feature. The temperature is high, and the remissions are not so marked. The characteristic features develop about the beginning of the second week. The tongue is then dry and furred. Anorexia is marked, and vomiting frequent. There is abdominal pain, meteorism, and diarrhœa. The stools are frequently pea soup in character, and have a strikingly offensive odour, somewhat resembling that emanating from the breath and from the purulent otorrhœa, if there be otitis media. The patient is affected with languor, muttering delirium, and great prostration. The face is pale and pinched, and a bright blush often mantles the cheek. The skin is occasionally affected with a rather dark measly rash, which does not disappear on pressure, and is not raised above the level of the skin.

With the exception of the rash, the condition of the patient so closely resembles that which is seen in enteric fever, that experienced physicians have attributed it to this disease. This is the more apt to occur as otorrhœa is either not observed, or when seen is regarded as a mere coincidence. In contradistinction to enteric fever there are no rose-coloured spots—though there are cases of undoubted enteric without such cuticular manifestations—and when a rash is present in infective thrombosis, it may be easily distinguished from the enteric rash by, its general appearance, the fact that it is not elevated above the skin, does not disappear on pressure, and does not come out in successive crops.

As a rule, the cephalalgia is much more persistent and severe than in enteric, the patient in the earlier stages of the disease complaining constantly of pain. The presence of infective otitis media and unilateral pain on pressure in the submastoid and sub-occipital regions, as also the thickening of the glandular and other soft tissues in those regions, and down the course of the internal jugular, materially aids in the differentiation.

The duration of those cases which exhibit symptoms of a markedly typhoid type, though subject to considerable variation, is somewhat shorter than in the preceding type.

The meningeal group of symptoms is much more seldom met with than either of the preceding, and is rarely found without association with one or other of the formerly mentioned groups. The meningitis may arise from the infective thrombosis, but it may also originate directly from the primary source of the disease, and appear as a complication, the symptoms of which may predominate over those of the sinus thrombosis. When lepto-meningitis occurs, the course of the case is more rapid than in the two preceding forms, generally lasting a week or a fortnight.

Cephalalgia is violent at the outset, and is often referred to the frontal region, though when otitis media is the primary disease, the pain may be most marked in the temporal. Vomiting is frequent. The temperature is persistently high. Rigors are not a feature of the meningeal form; they seldom occur at the outset, and when they appear in the second week, they indicate complications, such as infective absorption or purulent lepto-meningitis. There is great excitement, hypersensitiveness, and agitation. Certain groups of muscles, especially of the face, neck, and upper extremities, are subject to clonic and tonic contractions or to paresis. There may be contractions of one group of muscles and paresis of another, and both these are evanescent and fluctuating. Squint is common. The other cerebral symptoms depend on the special region of the brain affected. When the sigmoid sinus is involved, retraction of the head on the neck is usual, and occasionally it is drawn to one side. Delirium occurs at the later stages. When the lepto-meningitis is spinal as well as cerebral, there are spinal indications, especially, girdle pains of a severe shooting character, and absolute prostration. The mind is clear and the senses over acute at the beginning; there is no hesitancy in answering questions, though occasionally there is extreme irascibility displayed if the interrogatory is prolonged. The patient passes into a comatose state as the fatal termination approaches.

Although the symptoms evinced by infective thrombosis are described under three groups, and though typical cases of each do occur, it must not be understood that all or even the majority of cases of infective sinus thrombosis can be distinctly

relegated to one or other type, as they are most often mixed: occasionally symptoms of all three mingle together, and frequently those of the first two varieties are seen in the same case, one group of symptoms predominating at an early, the other at a later period of the disease. Regarding the meningeal form, it is well to bear in mind that symptoms very closely resembling it may arise from perforation of the tegmen tympani and subsequent lepto-meningitis. Further, abscess in the temporo-sphenoidal lobe may be found as an additional complication, which is generally accompanied by the pulmonary-typhoid groups of symptoms. Such abscesses occasionally originate secondarily to the infective thrombosis, as they appear to be of more recent origin than the thrombus in the sinus, but they may also arise as independent lesions. The symptoms of the infective thrombosis as a rule mask those of the cerebral or cerebellar abscess.

THROMBOSIS OF SPECIAL SINUSES.

Local lesions may excite inflammatory thrombosis in any of the sinuses. When there are enlargements or inflammatory action of the Pacchionian bodies, thrombosis of the longitudinal sinus will probably occur. A neoplasm occurring about the basi-sphenoid, especially in the sella Turcica, would produce a lesion of the cavernous sinus (see Case XLVIII.), while tubercular ostitis or cholesteatoma and infective lesions of the mastoid or petrous bones would probably implicate the sigmoid sinus.

Superior longitudinal sinus thrombosis.—It has already been noted that the superior longitudinal sinus is the one most frequently affected with marasmic thrombosis, but from its superficial position and its connection with the parietal, the diploic, and the emissary veins of Santorini, it is likewise liable to thrombosis originating both from traumatic and inflammatory causes. Traumatism may readily induce inflammatory thrombosis, when a portion of the vault of the cranium is driven into the sinus, thus suddenly arresting the flow of the blood. Erysipelas of the scalp involving the vertex, the parietal, or occipital regions, inflammatory action of the diploë, necrosis and ulceration of the skull, are prone to be followed by thrombosis of the longi-

tudinal sinus. Like all other sinuses, thrombosis may affect it secondarily by extension of a thrombus beginning in a sinus situated at a distance. Thus thrombosis may extend into the longitudinal sinus from the lateral. Orbital cellulitis, necrosis, and caries in the frontal region and orbital cavity, and in the upper ethmoidal bones, are apt to occasion thrombosis of the cavernous sinus, and, less frequently, they affect the anterior part of the longitudinal sinus.

Symptoms.—There are no pathognomonic symptoms of thrombosis of the longitudinal sinus; but œdema of the scalp and over-distension of the veins in the parietal and occipital regions and occasionally in the frontal, especially when the anterior part of the longitudinal sinus is involved, are fairly constant accompaniments. A slight degree of squint is frequently observed. In an infant the scalp over the anterior fontanelle is often very œdematous when the longitudinal sinus is involved. Epistaxis occurs frequently, and, as one would expect from the venous connection of the nose with the longitudinal sinus in the first years of life, it is much more common in infancy and childhood. Abscess of the outside of the skull has been known to be associated with longitudinal sinus thrombosis. Convulsions and post-convulsive paralysis occasionally occur from thrombosis of the longitudinal sinus.

CASE XLIV. *Marasmic thrombosis of longitudinal sinus. Précis of a case of a girl, aged seven years, admitted into Royal Infirmary, affected with thrombosis of the longitudinal sinus, caused by marasmus, following enteric fever.*

Patient (a girl of 18 years) had had a severe attack of enteric fever, with profuse diarrhœa, lasting over three weeks, when she became convulsed and lost consciousness. The convulsion was unilateral for the most part, though both sides were involved. When consciousness was restored it was found that the first side convulsed was paralysed. Both before and after this convulsion she had several attacks of epistaxis. She died from exhaustion. The longitudinal sinus and the middle cerebral veins were thrombosed, and the whole parietal lobe on the side opposite the paralysed extremities was converted into a mass of red softening.

CASE XLV. *Précis of a case of thrombosis of longitudinal sinus, probably infective, arising from cario-necrosis of skull.*

Case of a girl, **J. J.**, aged 10 years, in Royal Infirmary, affected with thrombosis of the longitudinal sinus, arising from inflammatory action dependent upon extensive cario-necrosis of the skull, probably syphilitic, but which appeared after enteric fever, and which was in a septic condition on admission. The posterior portions of both parietal bones were entirely removed and the upper part of the occipital. Three inches of the longitudinal sinus, about an inch from the torcular Herophili, were exposed, and while the anterior inch was soft and compressible, the posterior inch and a half was hard and solid. Masses of granulation tissue sprouted from the posterior part of the sinus, and from there spread over the exposed dura.

The parietal and frontal scalp veins were very prominent, and she had external strabismus of the right eyeball. She was dull mentally. Ultimately she recovered.

CASE XLVI. *Précis of a case of extensive thrombosis of longitudinal sinus, due to infective traumatism.*

Male, aged 45, admitted into Royal Infirmary with extensive crush of scalp over right side of head from vertex nearly to ear, the underlying bone being deeply ingrained with infective matter. It resulted in extensive sloughing of the scalp, necrosis of the greater part of the right parietal bone, with extradural abscess and thrombosis of the longitudinal sinus. Multiple trephinings had to be resorted to.

The symptoms of thrombosis of the longitudinal sinus were masked by the formation of the intracranial extradural abscess, and the extensive destruction of the scalp. The greater part of the necrosed parietal had to be removed, and the longitudinal sinus was found to be filled partly by blood clot and partly by granulation tissue. There was marked œdema of the scalp on the left side and on the forehead, and the veins of the scalp generally were greatly swollen. Part of this swelling and œdema were no doubt due to inflammatory action, though the occlusion of the sinus was the chief factor. Patient ultimately recovered, and was afterwards engaged at his usual employment.

CASE XLVII. *Précis of a probable case of thrombosis of longitudinal sinus from traumatism.*

L. S., a man, aged 54 years, received a compound depressed fracture of the skull, situated at the upper part of both parietals, behind the auriculo-bregmatic line, but the depression was principally on the right side. On the fragments being elevated the dura was found ruptured and the brain

infiltrated with extravasated blood, some of which lay free in the subdural space and was removed. About an inch from the longitudinal sinus a large vein which entered the sinus was found to have been bruised and felt rather hard. The bones were reimplanted.

During the first twelve hours patient complained of a dull headache, and had slight divergent strabismus and dilated pupil of the eye on same side as the lesion. The veins over the anterior and parietal regious were much distended. About thirteen hours subsequently he had a left-sided convulsion, the leg being the first involved, followed by the arm and then by the face, after which the right side was implicated, and he became unconscious for two hours. This state was followed by post-convulsive paralysis of left leg and arm and paresis of face, which lasted for four days and then gradually lessened, coincident with the lessening of the great distension of the scalp veins and the accompanying œdema.

From the great venous distension of the frontal and parietal scalp veins it is probable that partial occlusion of the longitudinal sinus ensued within the first twelve hours, either from the direct damage it had sustained or from extension of the thrombus from the bruised cerebral vein. When this occurred the veins in front of the block in the sinus would become engorged, and greater effusion and œdema into the brain at the damaged part probably occurred, occasioning the convulsion and the subsequent paralysis. The thrombus in the longitudinal sinus probably became gradually absorbed, when there was a re-establishment of the venous circulation, or this might have been brought about by the cerebral veins removing the blood from the longitudinal sinus to the basal sinuses.

CAVERNOUS SINUS THROMBOSIS.—The cavernous is more rarely affected by thrombosis than any other large sinus. It is seldom thrombosed by itself, other sinuses generally participating, especially the petrosals, the sigmoid, and lateral. Very frequently it becomes affected by the thrombic process extending, from the sigmoid sinus through the petrosals, or from the ophthalmic veins; in the one case it is affected from behind, in the other from the front. When thrombosis arises primarily in the cavernous sinus, it is generally the result of inflammation extended through the ophthalmic veins. Cavernous sinus thrombosis is frequently accompanied by basilar meningitis, and occasionally by purulent thrombosis of the ophthalmic veins and abscess in the orbital cavity. The superior ophthalmic vein is the one more frequently affected, though both are usually involved.

Ætiology.—Marasmic thrombosis affects the larger sinuses, and seldom implicates the cavernous sinus except secondarily, by extension from the lateral or longitudinal. There is no case on record of marasmic thrombosis occurring in the cavernous sinus alone, and

none where the marasmic thrombosis originated in that sinus. Traumatism, in the shape of fractures of the base of the skull in the immediate vicinity of the cavernous sinus, inducing lepto-meningitis or inflammatory action at the seat of fracture, has been known to occasion thrombosis of the cavernous sinus.[1] A blow on the head without fracture of the skull on one known occasion set up thrombosis of the cavernous sinus.[2]

The principal cause of cavernous sinus thrombosis is infective inflammation in the orbital cavity, passing backward through the ophthalmic veins, or possibly extension of the inflammation along the sheaths of the nerves, inducing meningitis. When the meningitis is infective, thrombosis is much more likely to follow. Erysipelas of the face or root of nose occasioning phlebitis of the anterior facial and ophthalmic veins,[3] infective inflammation in the buccal, nasal, and pharyngeal cavities, or ulceration of the tonsils and pharynx inducing phlebitis or thrombosis of the veins of the pterygoid plexus, may indirectly induce thrombosis of the cavernous sinus. Caries of the body of the sphenoid, causing infective meningitis, has led to thrombosis of the cavernous sinus, in the same way as infective caries of the mastoid leads to thrombosis of the sigmoid.

In the nasal cavities, infective processes generally—infective traumatisms, the sequelæ of tubercular and syphilitic ulceration of the pituitary mucous membrane, polypi and malignant tumours —have all been known to induce, directly or indirectly, thrombo-phlebitis of the cavernous sinus. It has also followed ablation of fibrous polypi of the nasal fossa, and on one occasion it was attributed to the retention of purulent secretion, by the blocking of the nasal passages by a fibro-sarcoma. Besides these, infective inflammation of any of the cavities leading to the nasal fossæ—the maxillary, frontal, ethmoidal and sphenoidal cells—may induce cavernous sinus thrombosis.

Infective inflammation in either maxilla, by setting up phlebitis in the coronary, facial, angular, or ophthalmic veins, may induce

[1] A boy sustained a compound fracture of the base of the skull from a blow on the head with a ruler. The basi-sphenoid was fractured and there was a wound in the pharynx. Meningitis and cavernous sinus thrombosis ensued.—Mr. Jordan Lloyd in the *Ophthalmological Review*, November, 1884.

[2] Mr. Hulke's case (Mr. Bowman's) which simulated orbital aneurysm.—*Ophthalmic Hospital Reports*, 1859.

[3] Billroth mentions a case where a carbuncle of the head produced infective inflammation in a vein which entered the orbit joining the ophthalmic, and so occasioned thrombosis of the cavernous sinus.

cavernous sinus thrombosis. Such inflammations affecting the pharynx and tonsils may reach the cavernous sinus, the former through the pterygoid plexus to which the pharyngeal veins pass, the latter by the pterygoid plexus or by the veins passing through the foramen ovale.

Cases are recorded of cavernous sinus thrombosis arising from alveolo-dental periostitis following the extraction of a wisdom tooth, and also from mal-evolution of a wisdom tooth inducing abscess of the pterygo-maxillary fossa.

Symptoms.—In considering the symptoms, it ought to be borne in mind that though one cavernous sinus is at the outset alone affected, yet the thrombus in more than half of the cases spreads through the circular to the cavernous sinus on the opposite side. The symptoms, though unilateral to start with, frequently become bilateral at a later stage. Not only so, but the side on which they began may be partially restored, while the other side becomes markedly affected. This alternation of the seat of symptoms is a diagnostic distinction between inflammations confined to the orbital cavity and cavernous sinus thrombosis.

The preliminary symptoms vary considerably, and they bear a relation to the direction in which the cavernous sinus has been invaded.

Pain is one of the preliminary signs when there has been intracranial invasion, such as may arise by tumour pressing upon the nerves in the vicinity of the sinus; cephalalgia occurs in various regions, sometimes in one or all of the branches of the first division of the fifth; supra- and infra-orbital pain of a neuralgic type is common, though pain may be complained of on the top of the head or in the occiput. There are, however, two groups of symptoms very frequently present—one depending upon venous obstruction, the other on paralysis due to pressure on the nerves surrounding the cavernous venous plexus. From the first there is exophthalmos, œdema of the eyelids and corresponding side of the root of the nose, and a degree of chemosis; while from the second there is ptosis, strabismus, and pupillary manifestations indicative of involvement of the third nerve.

The exophthalmos may be present to a very variable extent. It is due to intra-orbital venous congestion and distension of the tissues behind the eyeball. When due to inflammation

FIG. 53.—FACIAL EXPRESSION IN CAVERNOUS SINUS THROMBOSIS.

Exophthalmos, with internal strabismus œdema of right eyelids and corresponding side of nose in an early stage of thrombosis of cavernous sinus (Case LII.).

FIG. 54.—FACIAL EXPRESSION IN CAVERNOUS SINUS THROMBOSIS.

The same as above, a few days later. The œdema of the right eyelids, side of nose, brow, and face has greatly increased. The left eyelids are still free (Case LII.).

the exophthalmos may arise within a few days—occasionally twenty-four hours—and subsides to a marked extent when the collateral circulation is established. It is especially marked when there is ophthalmic phlebitis and retro-orbital abscess (see Case LII.). Œdema of the eyelids and chemosis frequently accompany the exophthalmos originating in thrombosis of the cavernous sinus, and they are due to the same causes as the exophthalmos.[1] "Choked disc" does sometimes arise from

FIG. 55.—FACIAL EXPRESSION IN CAVERNOUS SINUS THROMBOSIS.

The same patient, later stage. The right eyelids comparatively free from œdema, while the left have become affected to a very marked extent, the œdema extending over brow, face, and neck. Proptosis was also marked (Case LII.).

thrombosis of the cavernous sinus, though it is of course due immediately to obstruction of the retinal and ophthalmic vessels.

Besides these, swelling of an œdematous kind is occasionally found in the pharynx and tonsil on the same side, when the thrombosis in the cavernous sinus has extended into the pterygoid plexus, and possibly into the pharyngeal vessels. Œdema of the corresponding side of the face, due to obstruction of the facial vein, may be seen occasionally, arising from extension of the thrombosis, but may be also due to extraneous causes. In obstruction of the cavernous sinus, which has pursued

[1] In the four cases seen by the author the œdema of the eyelids was distinct along with the exophthalmos, and in all the others there was chemosis but in variable degrees.

a chronic course, enlargement of the frontal and facial veins may occur, due to the flow of venous blood being diverted through the orbito-facial anastomosis.

The nerve symptoms are very important in diagnosis. The second, third, fourth, and sixth nerves, as well as the first division of the fifth, may all be involved. The third is most often implicated, ptosis being one of the most constant symptoms. Occasionally it is difficult to make sure that ptosis due to pressure on the third nerve is really present, as the œdema is so great that distension and fixation of the upper eyelid occur, and could be entirely occasioned by the œdema without any of the nerves in the vicinity of the sinus being involved. Myosis is generally present at the outset, but as the pressure becomes greater stabile mydriasis ensues. External strabismus occasionally occurs, though the form of strabismus depends on whether the third or the sixth becomes first affected. When the case is advanced, ophthalmoplegia may be complete. There is defect of vision, and in advanced cases amaurosis from pressure on the optic nerve. The great proptosis with œdema of the eyelids render the testing of vision difficult and often imperfect. The pain produced by interference with the first division of the fifth is very variable, and has already been referred to.

While, at the outset, all the symptoms of the venous obstruction and interference with the nerves become gradually developed and then pronounced in one eye, as the case progresses they may affect the opposite eye, causing there the same symptoms and appearances as were present in the first. The second eye is involved rapidly; only twenty-four hours may elapse between the initial stage and the full development of these appearances. Coincident with the development of the appearances in the second eye, there may even be a subsidence of those in the eye first affected. The implication of the second eye is a sure indication of thrombic extension to the opposite cavernous sinus. This alternate involvement of the eyes when present is pathognomonic of this affection, but it does not always occur in thrombosis of the cavernous sinus. The bilateral and alternate involvement indicates, therefore, that the symptoms are due to thrombosis of the cavernous sinus and not merely to intra-orbital inflammation.[1]

[1] In two of the author's cases there was only very slight chemosis and œdema of the one eyeball, while the appearances were characteristic in the other. In the two others both eyeballs were alternately implicated.

When the sinus is invaded from the nose there has been generally a purulent discharge of old standing from the nostril. Besides these special localizing evidences all the general symptoms of sinus thrombosis—rigors, high temperatures, vomiting, and secondary complications are present.

The duration of the disease is very variable, from a few days to several months.[1] The great majority end fatally, and absolutely all those due to infective thrombosis do so. In one reported instance, there was pulsation in the orbital cavity along with the exophthalmos, which led to the diagnosis of intracranial or orbital aneurism, for which the carotid artery was ligatured.

Accompanying cavernous sinus thrombosis there is frequently basilar meningitis: occasionally, hemiplegia or monoplegia. In one or two of these latter cases hæmorrhage has been found on the surface and in the substance of the brain.

CASE XLVIII. *Case of cavernous sinus thrombosis, originating in tumour of base of skull.*

Mr. B., 66 years of age, had been healthy all his life, and was a very regular and quiet-living man. He was seen by the author in February, 1891, in consultation with Dr. Lapraik, who gave the following history of his patient's illness. He had been on a visit to Millport in April, 1890, where he attended a meeting in a crowded, overheated room. He then walked home in the cold, and had a chill which resulted in an attack of what was considered to be influenza. From that time (nine months prior to my visit) he continued to have a pain in the left side of the head, above the left eyebrow and over the left side of the brow, which was attributed to neuralgia. Along with this pain he had a sensation as if the left nostril were stuffed. Both these symptoms came on immediately after the attack, and they persisted until the end of the case, though they became merged into others subsequently developed. These symptoms continued for five months, at the end of which he caught a fresh cold. The pain then became intense. The sensation of cold in the front of the brow was marked, but was relieved some days later after a free discharge of foul-smelling pus from the left nostril. About the same time he felt great pain in the left eyeball, and the eyelids began to swell. He was seen by an oculist, Dr. Reid, who found that the eye was free from disease, though there was inflammation surrounding the eyeball.

When seen by author he had very marked swelling of the left eyelids, and slight exophthalmos of the left eyeball, which was fixed, and could not be rotated in any direction—complete ophthalmoplegia externa. The pupil was

[1] Mr. Bowman's patient lived five months, the author's nine months, though in the latter, the symptoms at first might have been due to the basal tumour.

dilated and fixed; there was amaurosis, though he could distinguish light. The eyelids were chemosed. The eyeball was very tender to touch. The left side of the face and nose, and the left side of the forehead, were swollen and discoloured from capillary injection; there was also œdema, which could be dispelled by firm pressure. The right eyeball was likewise affected, but to a much smaller extent than its neighbour. There was a slight swelling of the eyelids and chemosis. The pupil was slightly contracted and sluggish. Vision in that eye was good. There was slight convergent strabismus, and he could not rotate his eyeball outwards. The discharge from the left nostril was purulent and fœtid.

A diagnosis was given of cavernous sinus thrombosis, with abscess in the orbital cavity. It was probable that he also suffered from basal tumour, owing to the length of time he had experienced the preliminary symptoms, though the sudden manner in which the pains in the head and the attack of influenza came on rather favoured an inflammatory origin. Whatever was the origin of the thrombosis, it was clear that there was now a purulent collection, probably about the left orbit and in the cavernous sinus. Ablation of the contents of the left orbital cavity was advised, in the hope of affording an exit to the inflammatory secretion. This proposal was negatived by the friends, because the prospects of relief were doubtful. Two days afterwards he became affected by paralysis of the right side of the body, which lasted a few days, then passed off, leaving him, however, stupid and confused. The left eyelids became greatly swollen and chemosed, and the eyeball fixed, while the swelling of the right eyelids also increased so much that they could scarcely be opened, even by the application of firm pressure. To afford temporary relief the conjunctiva had several times to be snipped. Patient died at the end of a fortnight from the time of author's first seeing him, and over nine months from the commencement of his illness.

Post-Mortem Examination.—The *sectio* of his head showed abscess in the left orbital cavity, pus inside of the sheath of the optic nerve, thrombosis of the superior ophthalmic and many smaller veins in the orbital cavity, thrombosis of the cavernous sinus, intense lepto-meningitis, specially marked at the base of the brain on the left side, though the whole of the cerebral lobes were soldered. There was a tough, firm tumour in the left anterior part of the middle fossa on the wing of the sphenoid, which enclosed one of the vessels running from the cavernous sinus. This was a neoplasmic formation, and it was removed for further examination. There had been a slight purulent discharge from the left ear during the last week of his life, but there was no communication between the skull and the roof of the tympanum. It is probable that the inflammatory action from the nasal cavity, with its fœtid discharge, had found a way through the Eustachian tube to the middle ear. There was nothing more than a little discharge in the middle ear when it was opened. There was no bone erosion at that part. The membrane had a small perforation. The microscopic examination of the tumour showed it to be a carcinoma.

CASE XLIX. *Infective thrombosis of cavernous sinus following a pustule on the mucous membrane of the ala of the left nostril. Duration of illness six days. Came under observation on sixth day of illness.*

Mrs. D., aged 28 years. This patient was seen by author in consultation with Dr. J. Adams in private on 30th September, 1891, about six days from the beginning of her illness.

On 24th September when she had been in a warehouse inspecting dresses she felt shivery. That night she felt an itching in her nose. This irritation in the nostril increased, and a pustule formed on the inside of left ala. She was able, however, to go out and visit friends on the 26th inst. The same evening it got much worse. On the 27th she was visited by Dr. J. Adams, who incised the pustule in the nostril. On the 28th the nose was much swollen, and a red œdematous patch had formed about the inner canthus of the left eye. On that day her medical attendant made an incision about the neighbourhood of the orifice of the lachrymal sac, and relieved a small quantity of pus which had accumulated. On the 29th September, late at night, she became delirious. The left eyeball became swollen, and Dr. Reid, the oculist, was consulted; he at once said it was not an affection confined to the eyeball, and recommended that a surgeon should see it.

The patient was in the following condition when seen by author on the 30th September, on sixth day of illness:—

She was in an insensible condition, passing urine in bed; her face was greatly swollen, the forehead and both eyelids being most discoloured and œdematous. The left eyeball was markedly protruded. The whole of the left ocular muscles were paralysed, as the eyeball remained fixed while its fellow occasionally moved. The left pupil was in a state of stabile mydriasis, while the pupil on the right side was mobile and less than medium. The conjunctiva of the left eyeball was chemosed. Pulse was very rapid and extremely feeble. Temperature was 104°, and she had all the appearance of a person dying from infective systemic poisoning. The pustule on the left nostril had a hardened base, was whitish round the outside and had a dark centre; it was situated within the left ala, so that the whole of its circumference was rather difficult to see.

The diagnosis was that of thrombosis of the cavernous sinus of infective origin, the pustule in the nostril having been the primary focus from which the pathogenic organisms had spread, possibly along the course of the veins surrounding the nasal duct or into the left facial vein, then to the superior ophthalmic, and so into the cavernous sinus.

The patient was evidently dying, but in the hope of somewhat relieving her condition, the left eyeball was removed, the thrombosed vessels being cleared out, so that the pent-up matter about the sinus would have an opportunity of escaping. In the operation extremely little blood escaped, probably owing to the thrombosis of the ophthalmic veins and sinus. There was no abscess in the line of the superior ophthalmic, although the vessels were markedly thrombosed. Patient was so weak that chloroform

could not be given. As she was insensible she experienced no pain. The operation seemed to afford patient relief, but she never recovered consciousness. Her temperature increased to 106°. She had profuse diarrhœa; had an imperceptible pulse for some hours before death, which occurred about midnight of the same date.

Post-mortem examination was refused. Duration of illness six days.

CASE L. *Précis of a case of cavernous sinus thrombosis, due to infective traumatism of right orbit.*

G. J., aged eighteen years, was admitted into the Royal Infirmary, with thrombosis of the cavernous sinus, due to a penetrating wound of the right eyeball, a cane having been thrust through the eyeball fourteen days prior to admission. There was marked double exophthalmos, with complete fixation and chemosis, along with œdema of the eyelids. These were most marked on the injured side, where they had originated, and to which they were confined for the first ten days. The temperature was 105° to 106° F. The patient was unconscious and *in extremis* when admitted, and only lived fourteen hours thereafter. At the post-mortem examination there was found purulent phlebitis and retro-orbital abscess in the wounded orbit, the eyeball being disintegrated and pus being formed within the sheath of the optic nerve, and passing backwards into the brain. The veins in the left orbital cavity were much enlarged and inflamed. There was no pus in that cavity. The cavernous sinuses and circular sinus were all thrombosed; on the right side the thrombus was purulent. There was basilar meningitis, which was purulent in the vicinity of the sinus.

CASE LI. *Précis of a case of cavernous sinus thrombosis, from infective ozœna, arising from syphilitic caries.*

A man of forty-five years of age was seen by me unofficially in the wards of my late colleague, Dr. Scott-Orr, physician to the Royal Infirmary. He had suffered for many years from ozœna, due to syphilitic caries of the turbinated ethmoidal and sphenoidal bones, and was at time of my visit the subject of intracranial symptoms, evidently related to cavernous sinus thrombosis. The exophthalmos, with wide dilatation of the pupils, and complete fixation of both eyeballs; the œdema of both eyelids, forehead, and root of nose, coupled with intense cephalalgia, were marked features. The patient died some days afterwards, and the partial post-mortem which was obtained was reported to me as being in accordance with the above.

CASE LII. *Infective thrombosis of cavernous sinuses both right petrosal and right sigmoid sinuses, the ophthalmic, the posterior condylar, and the upper part of internal jugular veins, with traumatic pneumonia due to vagus implication, all arising from otitis media purulenta. Coming under observation on eighteenth day of illness. Duration of disease, thirty days.*

E. C., aged twenty, admitted into Royal Infirmary, 18th December, 1889, on eighteenth day of her illness, suffering from extensive thrombosis of the sigmoid and cavernous sinuses, meningitis, and traumatic pneumonia of the right lung, proceeding from right infective otitis media.

History.—*Heredity.*—Family fairly healthy, one brother having died with symptoms of tuberculosis of lung. *Personal.*—At an early period of her life she had disease of one of the bones of the right fore arm, the cicatrix of which is still evident. Her friends were not aware that she had any discharge from the ear, but a year ago she complained of slight deafness in the right ear, and says that at that time there was a slight otorrhœa; otherwise she has been fairly strong, healthy, and regularly at work.

Present Illness.—On 30th November, 1889, she complained of pain in the right ear, which came on suddenly, and after some hours extended all over the temporal region. It was accompanied by giddiness, which prevented her from walking. An aurist, who was consulted on 4th December, passed a Eustachian catheter and syringed the ear. On the afternoon of that day she became much worse, the pain extending to the vertex and back of neck. On the same evening she had a rigor, a second occurred on 6th December, and a third on the 7th. When she sat up vomiting was frequent. On 8th December the pain was worse, and principally affected the vertex and the back of the neck. She could scarcely raise her head from the pillow; her neck felt stiff, and the head was retracted. She also experienced pain in the throat.

On December 11th she was admitted to one of the medical wards of the Royal Infirmary, and was then complaining of intense pain in the ear and head. She was flushed, feverish, thirsty, but was quite conscious and intelligent. She had a temperature of 105° F., perspired profusely, and the same afternoon she had a fourth rigor. On the 12th she had slight congestion at the back of both lungs. Slight puffiness over the right upper eyelid was noted, and she experienced facial and frontal pain. Rigors occurred daily.

Condition on Admission.—She was transferred to ward 22, under Dr. Macewen's care, on 18th December, the eighteenth day of illness, and her condition during the first few subsequent days was as follows :—

The patient presented the appearance of a well-developed young woman, who had become pale, emaciated, and extremely feeble. She was quite conscious, and possessed clear, quick cerebration, though she was easily fatigued by continued interrogation. She complained of pain over the right side of head and neck, which was increased on movement. There was

tenderness on percussion over the temporal, parietal, and occipital regions. Acute pain was experienced when deep pressure was applied to the soft tissues on the upper half of the right side of the neck, especially acute and severe at the apex of the posterior cervical triangle and along the line of the internal jugular vein. Similar pressure over the left side of the neck did not cause pain. Dr. Johnstone and Dr. Pringle, house surgeons, both said that sharp downward pressure of the head, applied to the vertex in the long axis of the spine, also caused patient to wince. There was swelling and induration of the soft tissues in the upper third of the right side of the neck, most marked in the sub-mastoid region, but also presenting post-pharyngeally, possibly due to a distension of the pterygoid plexus, which receives from the cavernous sinus several direct branches along which the inflammatory action might have extended. There was also a prominence of the right tonsil. The upper third of the sterno-mastoid felt somewhat fused with the other soft tissues. This swelling was deeply seated, detectable on palpation, though presenting little external appearance. The right sub-mastoid and sub-occipital glands were enlarged, several being about the size of small grapes. She had pain in the throat, a difficulty in swallowing ; and it was feared that particles of fluid food entered the trachea, as she frequently coughed after attempting to swallow food. Her voice was husky, and she stated she had difficulty in speaking as her voice was "altered." Since her illness she also experienced a sensation of dyspnœa, had a slight, catching cough, and a rusty coloured expectoration. There was a diffuse pneumonia of both lungs, at first most marked on the right side, but it subsequently became general.

Motor Power over the body generally was fair, considering her enfeebled condition. There was no paralysis, except of the ocular muscles.

Sensations.—Cutaneous sensations were normal, with the exception of a hyper-sensibility of the upper half of right side of face and brow.

Reflexes.—Plantar reflexes very slight in both feet. Patellar reflex normal ; ankle clonus absent.

Eyes.—There was exophthalmos of right eyeball and ptosis of the right eyelid. Internal strabismus existed, with a little inclination upwards and inability to rotate eyeball outwards. The pupil was in a state of stabile mydriasis. There was diplopia, and when she wished to look at any object she kept the right eyelid closed. The left eye was normal. *Ophthalmoscopic examination.*—The right disc had a blurred outline, with a slight woolly appearance, giving the aspect of œdema. The left disc was slightly hyperæmic, due probably to hypermetropia, which she had to the extent of 3 D.

Ear.—There was a large perforation in the right membrana tympani, through which thick fœtid pus could be removed with a scoop from the tympanic cavity, but very little flowed therefrom. *Hearing.*—She was absolutely deaf to ærial and bone conduction on the right side. On the left she heard a watch about three inches distant. The temperature, pulse and respiration rates, were all high, the first sometimes reaching 106° F. Vomiting occasionally occurred, irrespective of food. Her bowels were constipated, and the urine was dark coloured, not albuminous.

Subsequent Condition.—The pneumonia gradually extended until both lungs were involved, but though the pneumonia was diffuse it was slight. The expectoration increased somewhat, and was prune juice in character, but latterly it became mixed with a good deal of frothy bronchitic expectoration. The odour from it was very slight and inoffensive.

In these particulars the pneumonia was by no means of the same severe character as that met with in the former cases where it arose from suppurative and disintegrating thrombosis of the lateral sinus leading directly to pulmonary infarction and subsequently to abscess in the lungs. This is well seen when one compares the notes of the former cases with this one. So marked was this difference that the question arose whether the pneumonia was traumatic, occasioned by the inhalation of particles of food, owing to the partial paralysis of the right vagus? The implication of the vagus might have occurred in either of two ways; by the thrombosed vein exercising pressure on the nerve as it passed through the jugular foramen, or by the nerve becoming involved in the inflammatory action, spreading from the infective thrombosis. The view that the vagus was involved was supported by the degree of paralysis of the pharynx and larynx which was present, and which became more apparent as the post-pharyngeal swelling disappeared, which it did a few days after admission into Royal Infirmary, and as it did so the dyspnœa and dysphagia increased. Tbe cough was slight and ineffective, so that she experienced difficulty in expectorating. She complained of diffuse pains at the lower part of the thorax and over the abdomen. These might have been due to irritation of the peripheral branches of the vagus, though they might have been occasioned by inflammation of the lungs and pleura. The expectoration was several times examined, and comparatively few organisms were found therein, whereas the expectoration from cases of infective infarction swarms with bacteria.

The exophthalmos of right eye increased, and the eyeball became fixed. There was complete amaurosis of that eye. There was extreme œdema of the upper eyelid, comparatively slight œdema of the ocular conjunctiva, though ultimately it became covered with a film of exudation, and emitted a profuse serous secretion which trickled over the cheek.

Along with this fixation and excessive protrusion of the eyeball the pupil became medium and fixed. There was hypersensitiveness of the parts surrounding the right orbit. The left eyeball at first suffered little with the exception of a slight paresis of the external rectus, and during one or two days œdema of the upper eyelid. Later, however, the left eyeball became involved almost to the same extent as the right, while at a still later stage, there was a subsidence of the proptosis in the right eyeball (see Figs. 53, 54, and 55). The vomiting continued very frequently, and bore no relation to the taking of food (milk). Rigors were frequent, she having had twelve during the time she was in Royal Infirmary (thirteen days in all); these twelve added to the ten which were previously recorded, make a total of twenty-two rigors.

She complained of a constant tiredness, a desire to sleep, and although she dosed a good deal, it apparently was not refreshing. In reviewing her

condition the conclusion arrived at was that she suffered from extensive thrombosis of the sinuses extending from the sigmoid and upper part of the internal jugular to the cavernous sinus and its tributary veins. From the high temperature and numerous rigors, it was believed this thrombus was in a state of disintegration, and though the lungs could have been involved by direct infective contamination, the evidence pointed rather to traumatic pneumonia produced through implication of the vagus.

In any view, the case was beyond the influence of surgical treatment. Patient gradually sank from extreme toxæmic prostration, her mind being

CHART M.—INFECTIVE THROMBOSIS OF CAVERNOUS SINUS.[1]

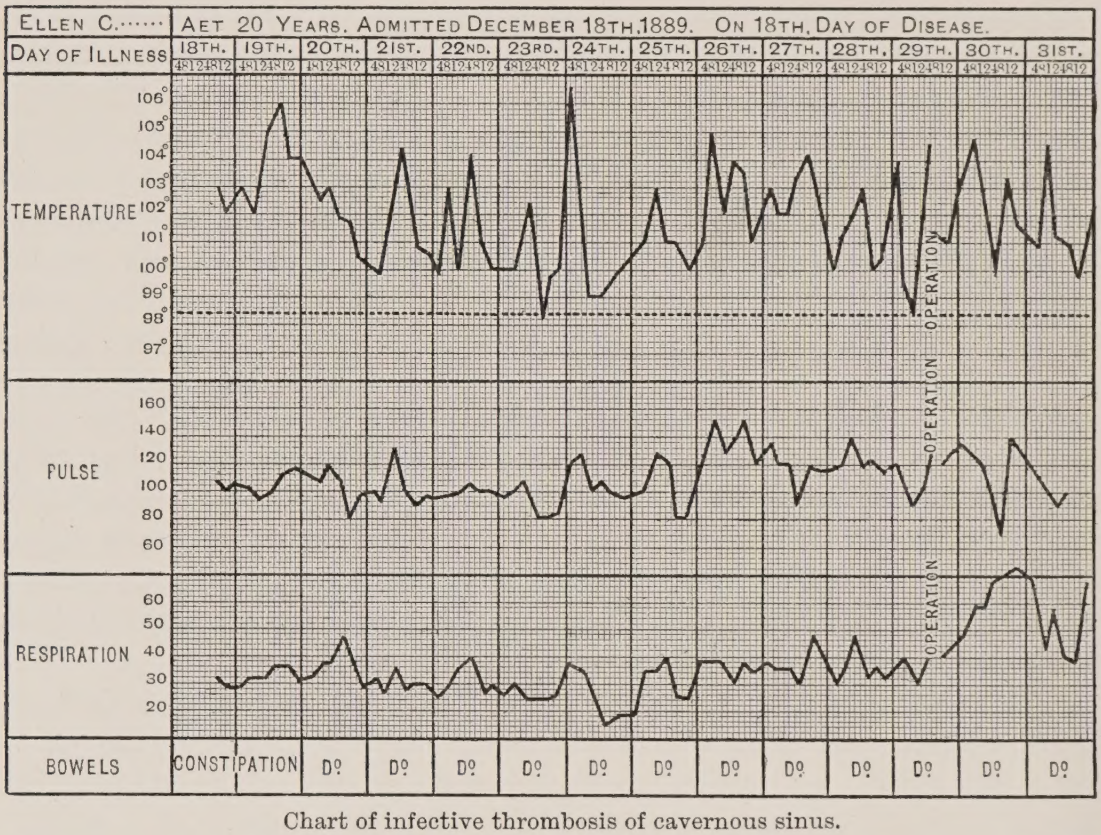

ELLEN C.·····
AET 20 YEARS. ADMITTED DECEMBER 18TH, 1889. ON 18TH, DAY OF DISEASE.
DAY OF ILLNESS
18TH. 19TH. 20TH. 21ST. 22ND. 23RD. 24TH. 25TH. 26TH. 27TH. 28TH. 29TH. 30TH. 31ST.
4 8 12 4 8 12 4 8 12 4 8 12 4 8 12 4 8 12 4 8 12 4 8 12 4 8 12 4 8 12 4 8 12 4 8 12 4 8 12
TEMPERATURE
106° 105° 104° 103° 102° 101° 100° 99° 98° 97°
PULSE
160 140 120 100 80 60
RESPIRATION
60 50 40 30 20
OPERATION
OPERATION
OPERATION
BOWELS
CONSTIPATION
Dº

Chart of infective thrombosis of cavernous sinus.

clear to the last. She died on the 30th December, 1889, on the thirtieth day of illness (see Temperature Chart).

Post-Mortem Examination.—The spinal cord was slightly congested otherwise normal. There was extensive external and intraventricular serous lepto-meningitis. Viewed from the inner aspect there was a thickening of the dura mater, on the right side over the tegmen tympani and along the right lateral and cavernous sinuses, which was of a dark brown colour with yellow patches. Between the right sigmoid sinus and the sigmoid groove was a slight exudation, and at parts, where the wall of the sinus had become softened and necrosed, there were several drops of pus. On the right side, the sigmoid, the cavernous, the inferior and superior petrosal sinuses, the superior ophthalmic vein, the internal jugular, and the anterior and posterior condylar veins, were all thrombosed.

[1] The operation referred to in the above Chart (as also in Chart D, p. 135), was only an incision over the eyelid to relieve the œdema.

The thrombi in the sigmoid and cavernous sinuses were disintegrated and partially converted into pus. The walls of these vessels next to the bone were softened, necrotic, and a few drops of pus lay between them and the bone. The bone in contact with the cavernous sinus and with one part of the sigmoid sinus was of a dark colour, and was slightly eroded.

The brain was in a fairly healthy condition, though the lining membrane of the ventricles was congested. The cerebellum had on its right side a dark stain corresponding to the position of the sinus. (The photograph, see Fig. 52, shows the mark of the sinus on the cerebellum.) All the structures passing through the jugular foramen were closely matted together by plastic effusion, and were more or less fused into one mass along with the soft tissues close to the right occipital condyle and the sub-mastoid region. On the outside of the skull continuous with this mass were a large number of lymphatic glands surrounding the thrombosed veins; all these tissues were deeply stained of a very dark greenish blue colour. In the lower part of the internal jugular there were small portions of thrombus adhering slightly to the coats of the vein, as if in process of dissemination.

In the orbital cavity, above and a little to the outer side of the right eyeball, there was an abscess closely connected with the superior ophthalmic vein, containing about half a drachm of pus. Otherwise the cellular tissue in the orbital cavity was œdematous.

Lungs.—The lungs were generally deeply congested and distinctly pneumonic at the bases. There was a good deal of pleuritic effusion in the right side, and in the base of the right lung there were several patches of a greyish colour, externally somewhat thickened, containing exudation, possibly purulent; these parts were removed for histological examination. Microscopical section showed the vessels in the pulmonary tissues to be deeply engorged, the spaces being filled with non-purulent exudation. The left lung was in much the same condition as the right; there was a little dry pleurisy; there were no infarctions or abscesses in the lungs. There was an excessive amount of fluid in the pericardium, but the heart was healthy. The spleen was a little enlarged and soft, the kidneys were anæmic.

Examination of Base of Skull.—The tegmen tympani was very much thinned, and perforated at four places, one along its outer aspect and three along its inner border; all of these were on the outer side of the ridge enclosing the superior semicircular canal. The groove for the sigmoid sinus was also eroded at the part where the temporal bone joins the occipital. This erosion was superficial, and the bone above and below this point was of a dark colour, having been stained by the thrombus in the sigmoid sinus. The staining and erosion were very marked in the jugular fossa. A similar superficial erosion was found in the course of the inferior petrosal, and especially over the seat of the cavernous sinus, where the basilar portion of the sphenoid was deeply eroded and likewise stained from the infective matter in the thrombosed sinus. A similar erosion of slighter extent existed outside of the skull, on the outer and anterior aspect of the right occipital condyle, and also on the corresponding portion of the atlas. This erosion was occasioned by the proximity of the infective thrombosis in the internal jugular,

and the state of these eroded cartilages and bones would account for the pain caused by the downward pressure of head against the vertebræ mentioned in report. All of these erosions seem as if they had been produced by contact with the infective products from the interior of the veins. The cortical parts of the bone were alone involved. This remark does not apply to the apertures in the tegmen tympani, which were distinct perforations from erosions originating within the tympanum.

THE SIGMOID SINUS.—The sigmoid is more frequently affected with thrombosis than any other sinus, lying as it does in such close proximity to the middle ear, the mastoid antrum and cells, all which are so frequently the seat of infective inflammation. The superior petrosal is so closely associated with the sigmoid that it frequently participates in the thrombosis of the latter; but occasionally the thrombosis originates in the petrosal and extends from it into the sigmoid. The minute veins of the antrum and middle ear partly pass into the sigmoid and partly into the petrosal; hence it is possible there may be two foci, one in each sinus, originating from the same source.

Sigmoid sinus thrombosis is rare in infants, occurs less seldom in children, is common in adolescent and young adult life, and is rare in old age.

In infants it is seldom seen owing to the following facts—first, the mastoid cells are not developed in them though the antrum exists; secondly, the squamous covering of the antrum is not yet soldered to the mastoid, and, therefore, purulent matter finds a ready exit, not being enclosed in a complete bony casing; thirdly, more numerous exits for the venous blood exist in infants, compared with adults; and fourthly, the sigmoid sinus rests on a flatter osseous surface than in adults—the bony gutter which imbeds the adult sinus being not yet fully formed. In infants, the internal ear is more exposed than in adults to pathogenic encroachments from the middle ear, hence in them leptomeningitis is apt to ensue which frequently ends fatally, and that so rapidly as to prevent the formation of sigmoid sinus thrombosis.

Ætiology.—Thrombosis of the sigmoid sinus may arise in a variety of ways, the following six being the principal:—(1) Extension from chronic purulent inflammation of the middle ear; (2) extension of acute inflammatory disease from the mouth, pharynx, and tonsils into the middle ear, antrum, and cells;[1] (3) extension of

[1] A case of otitis media of acute origin arising from an acute throat affection, probably diphtheria, was seen by the author in the Royal Infirmary, the whole duration of discharge from the external ear lasting only about

thrombosis from other sinuses; (4) traumatism such as fracture of the base passing from the middle ear to the sinus; (5) pressure of tumours; (6) infection extending from septic wounds or ulcers of the head, neck, or mastoid regions. The last three of these are rarely met with as causes of sigmoid sinus thrombosis. The tumour most likely to give rise to sigmoid sinus thrombosis is carcinoma of the middle ear, involving the mastoid and petrous bone, but it is questionable whether in every such case the thrombosis is due to pressure of the tumour, as it may also arise from the infective discharge which so often accompanies carcinomatous ulceration.[1]

Thrombosis does not frequently extend from other sinuses into the sigmoid except from the superior petrosal sinus. Marasmic thrombosis, met with at the extremes of life—the very young or very old—or in exhausting diseases, originating in the longitudinal sinus, occasionally extends along one or other lateral into the corresponding sigmoid. Acute diseases affecting the mouth, tonsils, and pharynx, especially those which produce or are accompanied by inflammatory action such as diphtheria and scarlet fever, sometimes extend along the Eustachian tube and cause acute suppurative inflammation in the middle ear, occasionally ending in thrombosis of the sigmoid sinus. Though usual, it is therefore not absolutely necessary to have osseous erosion in order to have sinus thrombosis, but there are very few cases without extensive bone disease.

By far the most common cause of sigmoid sinus thrombosis is extension of infective inflammation from the middle ear, either by direct osseous erosion or by the production of infective thrombosis in the minute veins passing from the middle ear to the sigmoid sinus. A purulent discharge from the middle ear may proceed for years, during which the bones are undergoing erosion, and the osseous plates separating the sigmoid groove from the antrum or middle ear are becoming thinned. The ulceration may

eight days prior to admission. The sinus was markedly thrombosed. Katz of Berlin had a case of diphtheria, followed by otitis media, which is given in *Deutsche Medicinische Wochenschrift*, No. 48. 1887. Zanfal (*Arch. für Ohrenheilkunde*, xvii. p. 157. 1881) mentions a case of thrombosis of the sigmoid sinus, arising from acute catarrh of the middle ear, in which there were pathogenic organisms in the sinus as well as in the blood clot.

[1] Of three cases of carcinoma of the middle ear extending into the petrous and mastoid bones, which have been seen by the author, one only had sigmoid sinus thrombosis. In it there was a profuse foul-smelling discharge, and the sinus was not directly pressed on by the tumour, though the sigmoid groove was perforated.

continue to progress slowly until the mastoid and even the petrous portion becomes a mere shell filled with granulation tissue. In such cases very little or no pus may be formed, a condition resembling caries sicca. Perforation of the thin osseous plates ultimately occurs and then the sigmoid groove is exposed. Through these openings granulation tissue, springing from the dura mater of the sinus, protrudes into the eroded apertures in the mastoid cells, antrum, or middle ear. As long as these parts are maintained aseptic, sinus thrombosis is unlikely to occur, except in a constructive form; but when pathogenic organisms are introduced, occasioning an acute inflammatory action, thrombosis is liable to take place.

In acute infective cases the minute veins of the middle ear and antrum may become thrombosed, and induce thrombosis of the sigmoid by thrombic extension. This may occur with little or no perceptible osseous disintegration.

Symptoms and appearances.—In a case of chronic otitis media, the cessation of otorrhœa coincident with accession of persistent otalgia extending into cephalalgia, high temperature with marked fluctuations, vomiting, and rigors, ought to be regarded as pointing in the direction of thrombosis of the sigmoid sinus.

The appearances accompanying sigmoid sinus thrombosis may be looked for from three sources. Those due to venous obstruction of the sinus, and consequent over-distension of the superficial veins, those resulting from inflammation of the mastoid, and those arising from inflammation of the veins communicating with the sinus.

In addition to these, evidence has been sought for in enlargement of the cervical glands, discriminating those which became enlarged on deep-seated irritation about the base of skull from those which enlarge from superficial irritation. While in a few instances, on putting this to the test, the glandular enlargement fairly corresponded to the above anatomical relations, and therefore tended to support the diagnosis, in many, there was no perceptible glandular increase at a period when such an enlargement would have been of diagnostic value, and in not a few cases, both superficial and deep cervical were apparently equally involved. As far as the evidence goes at present, glandular enlargement cannot be relied on as affording discriminating diagnostic data.

The appearances due to obstruction of the sinus and over-distension of the superficial veins are subject to considerable variation, are never obtrusive, and may be altogether negative. When present, the mastoid vein connecting the upper part of the sigmoid sinus with the external venous circulation is sometimes over-distended, and this over-distension is communicated to the vein which it joins on the outside of the skull. It will be at once apparent that the over-distension of these veins will be dependent on the extent and position of the thrombus in the sigmoid sinus. If this involves its upper part, then the internal orifice of the mastoid vein must be included in the thrombus, and the appearance of the vein externally will be normal. The deeply seated condylar veins assist in removing the blood from the lower part of the sinus, and their over-distension is entirely hidden during life. Regarding the increase in size of the veins on the opposite side of the head, no doubt a greater quantity of blood must pass through them when their fellows are thrombosed, but owing to their deep situation it is often difficult to decide whether they are abnormally dilated. In only a few instances of sigmoid sinus thrombosis were the appearances such as to enable one to conclude without hesitation that the veins were enlarged on the side opposite the thrombosed sinus.

Œdema of the mastoid region is described as an appearance which indicates thrombosis of the sigmoid sinus. Œdema of the superficial tissues over the mastoid does occur probably in about one half of the cases in which the sigmoid sinus is thrombosed; but œdema of this region occurs in many instances in which the sigmoid sinus is not involved in any pathological change, the œdema being due to inflammation of the mastoid process, especially its superficial parts. It is often seen in children affected with inflammation of the mastoid without sinus thrombosis. The œdema which occurs when thrombosis of the sinus exists is chiefly due to the ostitis of the mastoid and the blocking of the veins, though the occlusion of the sinus tends to augment this œdema. When the mastoid is eburnated, the sigmoid may be thrombosed without external œdema. When œdema is present in sigmoid sinus thrombosis, it is usually slight, extending from behind the auricle to the posterior aspect of the mastoid, and to a point somewhat below the level of its tip. The swelling which accompanies acute periostitis of the mastoid is so great, especially when abscess

has formed, that it cannot be confounded with the œdema here referred to (see three illustrated cases of acute periostitis of the mastoid).

Pain on percussion over the mastoid process is frequently elicited in cases where the sigmoid is involved, but it rather indicates deep mastoid inflammation. Pain is not produced on pressure over the mastoid, except when the superficial parts of the bone or periosteum are inflamed.

Phlebitis and thrombosis of veins.—Inflammation of the veins leading from the sigmoid sinus is a much more constant accompaniment of infective thrombosis. Phlebitis or thrombosis of the venous channels more or less closely associated with the sigmoid, especially of the internal jugular, the condylar, and the deep veins of the neck, into which the condylar veins empty themselves, and occasionally of the mastoid vein, affords indications which are of use in diagnosis. There are two areas of the neck where evidence may be sought—along the course of the internal jugular and in the upper third of the posterior cervical triangle. The symptoms are both subjective and objective. When the veins in these regions are inflamed, pain on pressure may be elicited even when the patient is deeply somnolent or semi-conscious. In the majority of cases of infective sinus thrombosis pressure on the internal jugular, especially on its upper third, causes acute pain. Testing with the same degree of pressure applied to both sides of the neck, the healthy and the diseased, enables one to compare the responses given by each. Occasionally, the application of this pain test has strengthened the evidence regarding the side affected, when this was deficient from other sources. It has also occurred that pain has been experienced on pressure on both sides, though much more severely on the one diseased. It must be remembered that infective phlebitis may spread to the opposite side, through the various connecting venous channels, so that both internal jugulars may be affected with phlebitis by extension from one source, though when this occurs the side in which the disease originated is usually much more deeply affected. In not a few cases, deep pressure applied to the upper third of the posterior cervical triangle causes pain. The phlebitis extending through the posterior and anterior condylar, and probably some of the basilar veins, affects the deep posterior cervical plexus and the occipital veins. The evidence of phle-

bitis in this region, owing to the depth of the veins, is obtained at a later period than that of the internal jugular, though in one instance, the veins at the upper third of the posterior cervical triangle were markedly inflamed, while the internal jugular remained unaffected till a late period, and then became painful only to a slight extent.

Thrombosis of the internal jugular when marked is very easy of detection as it lies so superficially. The finger perceives a cord-like formation to the inner side of the sterno-mastoid on the outer side of the artery, though the latter is sometimes overlapped by it. This may extend the whole length of the internal jugular, but it is frequently confined to the upper third. When thrombic disintegration has occurred, this cord-like formation may hardly be appreciable by digital examination, the walls of the vein alone being thickened. The veins in the upper third of the posterior cervical triangle when thrombosed are more difficult to detect owing to their comparatively small size, and the depth of their situation, but a degree of brawny swelling may generally be made out.

Enlargement of cervical glands and soft tissues.—At a later stage, as a consequence of infective thrombosis in the cervical region, the tissues in the vicinity of the bloodvessels become œdematous and infiltrated with plastic exudation, so that vessels, muscles, enlarged glands, and soft tissues generally, become fused into one mass. This infiltration may terminate in resolution, provided the source of contamination be removed, but if not, it will undergo purulent disintegration (see Case LIX.).

Cervical abscess arising from venous thrombosis.—Abscesses may form without any marked appearance of preliminary plastic infiltration; the cells poured out rapidly degenerate into pus. These abscesses may be seated either in the deeper parts of the upper third of the posterior cervical triangle in connection with the deep posterior cervical plexus, the vertebral plexus, and the occipital veins, or under the sterno-mastoid muscle in course of the internal jugular vein—always under the deep cervical fascia. In some instances they are in direct communication with the disintegrated thrombus in the interior of the veins. When these intravenous abscesses do form in the neck, the portions of the veins below are occluded with a firm thrombus, which, though exposed and liable to the same disintegration as that which overtook the upper part of the thrombus, is yet frequently suffi-

ciently firm to form a barrier between the lungs and the abscess. In one sense, therefore, the formation of such an abscess is favourable, as it shows that for the time there is a localization of the process of disintegration. It should be borne in mind that abscesses may form under the deep cervical fascia which, originating in middle ear disease, have direct connection with the mastoid cells through an aperture on the inner side of the mastoid process (see Case LX.).

Systemic affection—pulmonary involvement.—Much more formidable is the disintegration of the entire volume of the infective thrombus, the particles of it being carried by the blood into the lungs, where they set up infective infarction. It must be borne in mind that such depositions of infective particles may set up pulmonary infarctions, which at an early stage may be present in the lungs without giving rise to physical signs. The infective particles are conveyed to the lungs through the internal jugular, but may also be carried by the veins passing into the posterior cervical triangle, which flow through the vertebral and other channels to the subclavian.

Following the formation of infarctions in the lungs, the occurrence of fresh purulent foci is heralded by rigors, increase of the already high temperature, and profuse perspiration. The mind remains clear to the last, even when the thrombosis is accompanied by meningitis; though the patient may be fretful and irritable and his answers short, his comprehension is generally quick and clear. The symptoms of meningitis may mask those of thrombosis of the sinus.

Infective enteritis.—It is especially in thrombosis of the sigmoid sinus that the typhoid type of symptoms is apt to develop, and then the case is liable to be diagnosed as enteric fever. When diarrhœa is a marked feature, the stools have an intensified odour of the same character as that emitted from the purulent otorrhœa, the expectoration, and the breath. The patient often complains of a 'nasty' taste in the mouth, and the breath emits a marked putrid odour.

It is quite possible that purulent infective matter may find its way from the middle ear through the Eustachian tube into the pharynx, and so into the stomach and intestines, occasioning there the diarrhœa. The taste in the mouth perceived by the patient at an early period, both before the formation of the infarctions, and before the appearance of the diarrhœa, lends

colour to this possibility. Some of the infective secretion from the lung may also be swallowed, occasioning fresh irritation, or adding to that already established. It is difficult to prove in any given case that the infective enteritis arises in this way, as the general toxæmia of itself is quite sufficient to produce the enteritis. In one instance a patient was found to have from the pharynx a purulent secretion, which he had been swallowing, but which he was taught to spit out. It had the same odour as the pus from the ear. It was seen by posterior rhinoscopy to emanate from the position of the orifice of the Eustachian tube. It is also possible that a portion of this purulent secretion may be inspired into the lungs, where it may set up infective pneumonia.

It is important to note that partial thrombosis of the sigmoid may exist without sufficient symptoms to enable a positive diagnosis to be formed. A persistent purulent otorrhœa, co-existing with slight elevation of temperature, feeling of malaise, want of appetite, and slight sickness, may be all the manifestations detectable, yet on exposing the mastoid antrum and cells one finds the sigmoid groove already perforated by masses of granulation tissue adhering and sprouting from the anterior wall of the sinus, the lumen of which has been obliterated by clot and granulation tissue. In a few cases of this description, in which there was no thrombosis of the internal jugular, and only slight tenderness over its upper third, but where there was persistent infective otorrhœa, with increased temperature, the sinus was found partly thrombosed, and in one instance surrounded by purulent secretion, the mastoid antrum and cells being filled with pus and granulation tissue. In one such case there were slight moist râles over the lungs, which had persisted for at least a couple of months notwithstanding pulmonary treatment, but which disappeared after ablation of the mastoid along with about an inch of the thrombosed sinus.

The differential diagnosis, between infective sigmoid sinus thrombosis, cerebral abscess, and meningitis, is fairly easy in uncomplicated cases, but one must remember that all three may be present in the same case. Meningitis is frequently an accompaniment of advanced infective sigmoid sinus thrombosis, and it may assume a purulent form, spreading over the basilar portion of the brain and involving the medulla and cord. In the latter case retraction of the head and neck and muscular jerkings are apparent. The symptoms of infective sinus thrombosis generally mask those

of the secondary brain implication—of small cerebral or cerebellar abscess completely, and of meningitis partially (see Charts of Temperature, Pulse, and Respiration).

Prognosis.—The prognosis of sinus thrombosis may be regarded as always serious.

Marasmic thrombosis, when confined to a single sinus and when uncomplicated, is in itself by no means a fatal lesion, the gravity depending rather on the lowered constitutional condition which produced it than on the local result. Should the patient's general condition improve, the marasmic thrombosis may undergo absorption, when the lumen of the blood channel may be partially or wholly restored. Ramollissement of the brain, when it occurs owing to the occlusion of the cerebral tributary veins, renders the progress more serious; for even if life be preserved, more or less cerebral destruction is probable. Were the marasmic thrombosis to extend from the longitudinal through both laterals and sigmoid sinuses into the jugulars, a fatal result would supervene. Inflammatory infective thrombosis is much more serious, and more liable to end fatally. The great majority of cases of infective thrombosis, allowed to run their course without treatment, end in speedy death. At post-mortem examinations, however, there are occasionally found instances of long-standing obliteration of the internal jugular and sigmoid sinus, in connection with infective caries and extensive disintegration in the temporal bone.[1]

In such instances, it is probable that the pathogenic organisms inducing the inflammation have been so attenuated as to permit a formative process to occur in the thrombus; or the extension of the inflammation has been so gradual, and has been accompanied by granulation tissue, which slowly invaded the soft tissues, and by-and-by formed an obliterating connective tissue barrier within the sinus. The prognosis depends somewhat on the rapidity of the disintegrating process. If it be acute, infective infarction and lung disintegration are apt to follow, ending in speedy dissolution. Cases treated early, by thorough removal of the source of contamination from the sinus and its vicinity, offer a reasonable prospect of recovery. If the treatment be delayed until the thrombus has become disintegrated, and the blood contaminated with infective particles, which become lodged in the

[1] P. Hewett—*Lancet*, 1861—reports a case in which the lungs were apparently involved, yet recovery took place.

lung in the form of infarctions, treatment will then offer but a very distant hope. Still such cases ought to be operated on, as in several instances which exhibited all the symptoms of general infection at an early period, recovery took place after extensive operation and ablation of the source of infection.

CASE LIII. *Thrombosis of the right sigmoid sinus and right internal jugular vein, arising during an acute throat affection, probably diphtheritic. Recovery.*

A. O., 24 years, was admitted to Royal Infirmary on 1st Dec., 1890, in the second week of her illness, suffering from sigmoid sinus thrombosis, meningitis, and acute purulent otitis media, apparently originating from pharyngeal diphtheria.

Her family history and her previous personal history were good.

From the reports received from Drs. Allan and Davidson, of Belvidere, supplemented subsequently by patient's own statement, it appeared that she had been feeling out of sorts for a week or ten days previous to the beginning of the attack. She afterwards complained of headache and sore throat. During this period she had continued her duties as a nurse. She was then confined to bed with headache, and occasional vomiting and vertigo, which lasted during the first three days. On the fourth day, these symptoms increased, and in addition she became highly feverish, had stiffness of the muscles of the neck, pain behind right ear, and marked vertigo on attempted movement. The tongue was thickly coated with white fur. There appeared for the first time a slight discharge from the right ear. The patient herself, her mother, and near relations all declare that she never had discharge from either of her ears previously. This otorrhœa gradually increased in quantity, became distinctly purulent, and had a heavy odour which was subdued by boracic acid injections.

Condition on Admission.—When admitted into the surgical wards she was fretful, easily disturbed, and tossed about as if in pain. The pupils were unequal, the right being contracted and sluggish; occasionally there was slight external strabismus. Her visual field was good. There was no optic neuritis, though there was slight congestion of the disc. There was slight swelling over the upper third of the internal jugular, which was distinctly painful on pressure. Her pharynx and tonsils were intensely inflamed, especially the right tonsil, and subsequently these parts were covered by patches of greyish ash-coloured exudation. The temperature fluctuated between 103° and 105° F. There was no distinct paralysis. There was a purulent odourless discharge from the ear. The membrana tympani was inflamed and perforated. She heard a watch about four inches from the right ear and at least three feet from the left. There were slight râles over the chest, but no evidence of pulmonary infarction.

Operation (3rd Dec., 1890).—The mastoid antrum and cells, the middle ear and sigmoid sinus were exposed. Some drachms of pus escaped from the

antrum and middle ear. The thin plate of bone separating the cells from the sigmoid groove was removed, exposing the sinus. The sinus was in part covered by granulation tissue, apparently newly formed, and in part by a plastic layer containing pus. The sinus was hard and unyielding, its contents having become thrombosed, but there was as yet no evidence of disintegration. There was acute inflammation in the mucous membrane lining the antrum, which, though thus inflamed and softened, was yet intact, except toward the sigmoid groove, where it was converted into granulation tissue. The pus was removed and the sinus obliterated at this point. There was no disintegration in the thrombus.

Subsequent Course.—For the first few days after the operation patient seemed relieved, though the temperature still remained high. At the end of the second week the swelling in the upper third of the neck increased markedly till it reached the size of a hen's egg. It was principally confined to the internal jugular, though in addition there were a few enlarged glands. At one period the swelling was so great and suspiciously soft at one part that ligature of the internal jugular below that point and evacuation of the contents was determined on, but was delayed from the fact that below that place as far as could be reached by the finger, nearly to the junction of the internal jugular with subclavian, the internal jugular was filled with a firm dense cord-like mass, which stood out like a thick pencil, was not painful to touch, and formed a barrier between the disintegrating portion and the lungs. The lungs had meanwhile improved. A delay of forty-eight hours permitted a marked improvement to be discernible, and slowly during the three ensuing weeks the swelling of the internal jugular became absorbed. As the swelling receded, the temperature fell. The general condition of the patient subsequent to the operation was far from satisfactory. She became extremely weak mentally and physically, wandered while awake, and muttered while she dozed. She could not turn herself in bed, and though she had no actual paralysis, her muscular power was defective, and she swallowed fluids in small quantities with great difficulty.

Six weeks subsequent to the operation she was fairly convalescent. The wound had healed from the bottom by granulation tissue. She was dismissed well on the seventh week. At that time there was still plainly discernible a thick cord in line of the internal jugular.

Remarks.—The facts of this case appear to indicate that the acute throat affection had extended through the Eustachian tube to the middle ear, mastoid antrum, mastoid cells, sigmoid sinus, and probably to the meninges, over the tegmen tympani. From the greyish patches on the tonsils, especially the right, the intense inflammation of the pharynx and uvula, and the subsequent prostration and the marked post-pharyngeal paresis, the case was diagnosed as probably one of diphtheria. The layer of bone between the sigmoid sinus and the mastoid antrum and cells was very thin, the thinness not being due to disease, but to anatomical construction. The acute affection could therefore travel with greater ease in that direction.

CASE LIV. *Infective sigmoid sinus thrombosis, inducing the pulmonary meningeal group of symptoms, arising from left purulent otitis media. There were purulent pachy-meningitis, serous lepto-meningitis, infarctions, and pulmonary abscess, with disintegration. Death. Post-mortem. Admitted on eighth day of illness. Duration of illness twenty-four days.*

W. Y., male, æt. 7 years, was admitted into the Children's Hospital on 11th February, 1889, on the eighth day of his illness, suffering from infective otitis media of left side, accompanied by brain symptoms and probable lung mischief.

History.—*Heredity.*—The patient's parents were both healthy; they had three other children, all of whom are robust. *Personal.*—Patient had the ordinary children's ailments.

Present Illness.—Since he was a year old he has had left otorrhœa, but was able to attend school, to play, and to enjoy himself with the other children until one week prior to his admission into hospital, though shortly before this date his father observed that patient had delay in passing urine. On 4th February, seven days prior to admission, patient became ill, complaining of intense pain in the left ear and same side of head. The following day he was in no way relieved, and in addition he had a severe rigor. The rigors recurred on the third, fifth, and seventh days of his illness. On the last date the rigor was preceded by vomiting, and when it subsided the child took, without cause apparent to the parents, a screaming fit, which lasted twenty minutes and was followed by profuse perspiration. The pain in the head was constant from the onset of the attack, though subject to exacerbations, and when very severe parents believed he could not see.

Condition on Admission.—The appearance patient presented on admission was that of a well-developed, fairly-nourished boy, having flushed face and anxious aspect. He was intelligent, answered questions clearly, but soon got fatigued, when he became irritable and wished to be left alone. Though he remained quiet he was not drowsy. Sometimes he screeched just as patients do in acute lepto-meningitis. He complained of pain on the top of the head. His temperature was 103° to 104° F., his pulse quick, never under 100 and more often 120 per minute. He had an occasional very slight, catching, irritable cough, which might have been easily overlooked. It was similar to the cough present at an early period in Case No. LVII., and as in that case, when taken in conjunction with other symptoms, indicated a probable lung involvement, though as yet there were no abnormal physical signs. He urinated very frequently, but the quantity passed in twenty-four hours was normal, as were likewise the specific gravity and chemical reaction of the urine. Bowels were loose.

There was no paralysis. There was a free discharge of foul-smelling pus from the left ear. On aural examination granulation masses were seen to surround a perforated and eroded tympanic membrane. There was pain on pressure over the upper thirds of the internal jugular and posterior cervical triangle. His pupils were equal and responded to light. All the ocular muscles were active.

Ophthalmoscopic Examination showed congestion of both fundi, the vessels being engorged and the optic discs slightly blurred.

Subsequent Course of Disease.—Afterwards patient continued in much the same state, rigors occurring every second or third day. The rigors were generally preceded by flushing of the left side of the face, which during the shivering turned of an earthy pallor. The duration of the rigors was from ten to thirty minutes. His lungs were daily examined, and gave no physical indications of disease. On the 13th February he complained of pain of an acute catching character over the right side of the chest. On that date the lower two-thirds of the right lung was found slightly congested, the left lung as yet showing no sign of implication. His breathing became more rapid and irregular, the cough more frequent, louder, and deeper, and it occasioned him pain. It was evident that patient was suffering from lepto-meningitis, general systemic infection, and pulmonary inflammation, secondary to otitis media, probably communicated through the bloodvessels, especially the sigmoid sinus.

Remarks.—The possibility of direct contamination of the lungs by septic matter entering the trachea by way of the Eustachian tube and pharynx suggested itself, as it was found that he vomited purulent secretion which had the same odour as the discharge from the ear, and nurse believed she had seen patient swallowing his expectoration. If pus from the Eustachian tube found its way into the œsophagus it was possible for it to gain access to the lungs through the larynx and trachea. From the extreme tenderness along the course of the internal jugular on the left side of the neck evinced on pressure, it was probable that there was inflammation of the veins leading from and continuous with the sigmoid sinus, and therefore probable that the pathogenic organisms had been conveyed to the lungs through the blood-vessels. The head symptoms were those of acute meningitis. There were no indications of cerebral abscess.

Operation (13th February, on tenth day of illness).—The system having already become involved in the infective process, secondary deposits having occurred in the lungs, and the weakness of the patient being marked, it was resolved to confine the operation to the removal of the primary source of the disease in the middle ear, antrum, and its extension toward the sigmoid groove.

The mastoid antrum when opened was found to be enlarged, eroded, and filled with fœtid pus. The sigmoid sinus was exposed, and only a very thin layer of pus was found in the groove. The wall of the sigmoid was inflamed and thickened. These parts were thoroughly cleansed. The middle ear was cleared out, and a stream of fluid was passed from the antrum to the external ear.

During the time patient was under the influence of the anæsthetic the pulse did not abate its rapidity, continuing at the rate of 120 per minute, just as before the administration of the chloroform.

For eighteen hours after the operation there was a lull in the symptoms, but at the expiry of twenty-four hours the temperature, pulse, and respiration all rose to their former level. The pain in the right side of chest

increased, the right lung being still the only one affected. (In this unilateral implication of the lung it differed from Case No. LXVII, where both lungs were equally implicated from the outset.) The expectoration now became distinctive; it was prune juice in colour, and foul-smelling. Under the microscope it was seen to consist for the most part of pus and blood cells, and swarmed with bacteria. This expectoration rapidly increased, the odour becoming daily more offensive. The left lung became involved about a week later. The odour then resembled that emanating from gangrene of the lung.

CHART N.—INFECTIVE SIGMOID SINUS THROMBOSIS.

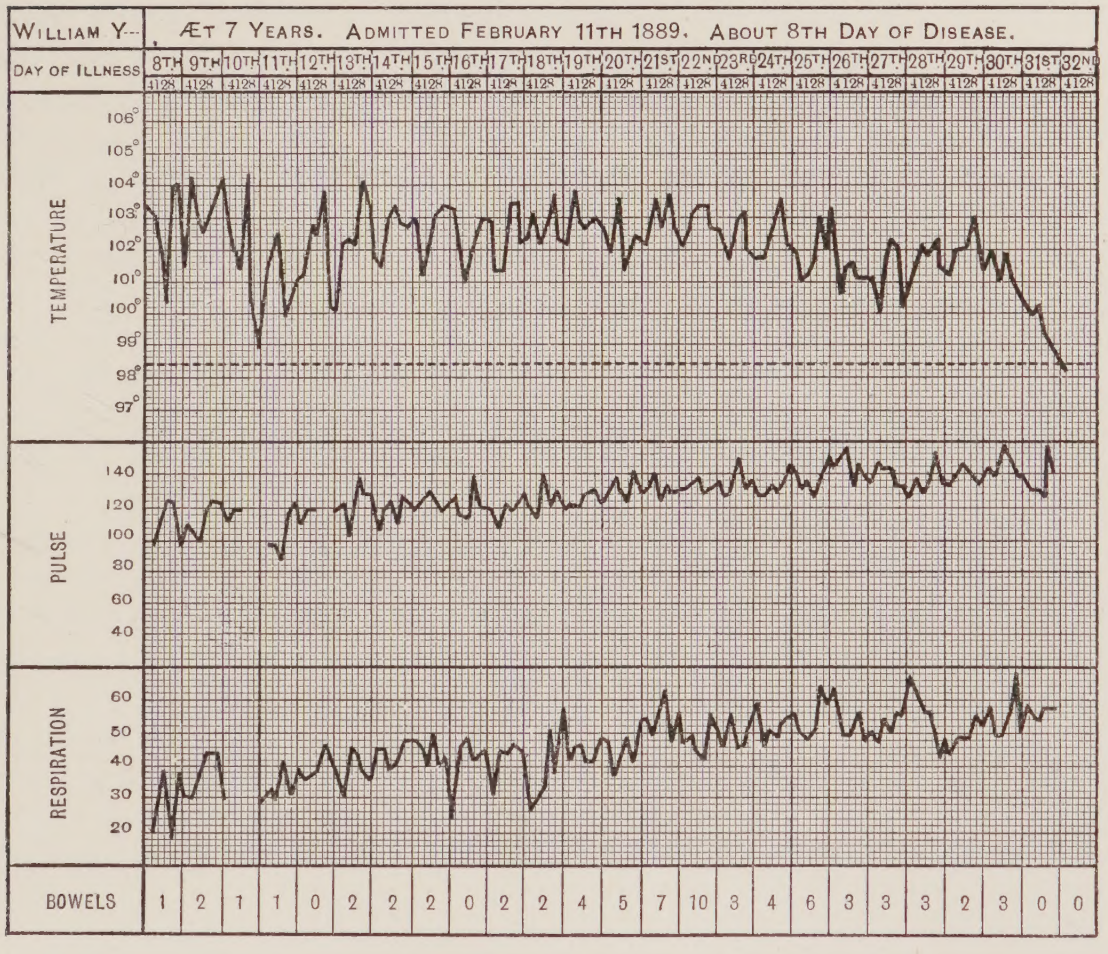

WILLIAM Y--
ÆT 7 YEARS. ADMITTED FEBRUARY 11TH 1889. ABOUT 8TH DAY OF DISEASE.
DAY OF ILLNESS
8TH 9TH 10TH 11TH 12TH 13TH 14TH 15TH 16TH 17TH 18TH 19TH 20TH 21ST 22ND 23RD 24TH 25TH 26TH 27TH 28TH 29TH 30TH 31ST 32ND
4 P.M.
TEMPERATURE
106° 105° 104° 103° 102° 101° 100° 99° 98° 97°
PULSE
140 120 100 80 60 40
RESPIRATION
60 50 40 30 20
BOWELS
1 2 1 1 0 2 2 2 0 2 2 4 5 7 10 3 4 6 3 3 3 2 3 0 0

Chart of infective sigmoid sinus thrombosis.

The patient had to be somewhat isolated, and the vessel containing the expectoration frequently cleansed and deodorized to prevent the other patients from being annoyed. The bowels were free from the date of admission into hospital. Distinct diarrhœa ultimately occurred. There was great irritability about bladder and rectum. Patient became very irritable, often screeching for many minutes consecutively. He had marked photophobia. Occasionally there was a slight squint in the left eye, the pupils were usually unequal, the left being as a rule the larger, though occasionally they were equal. They contracted sluggishly to light. Towards the end both were dilated and fixed. These symptoms were indicative of meningitis. The

joints were occasionally painful, especially the knees. Attacks of coughing were very troublesome, pus coming up in mouthfuls. The attacks were followed by profuse sweating. Patient became greatly emaciated and exhausted, and died comatose on the 7th March, the whole illness lasting twenty-four days. The chart giving the record of the temperature, pulse, and respiration taken every few hours during the whole course of intra-hospital life is striking and instructive.

Post-Mortem Examination.—The dura over the left petrous bone was thickened and congested. There was serous lepto-meningitis over the whole cerebrum and also partly over the cerebellum and basal ganglia. Over the left petrous bone and radiating outwards, forwards, as well as backwards from it, the pia mater was intensely congested. There was a very small quantity of extradural pus over the left petrous bone, a thin layer over the tegmen, and a similar layer lining the sigmoid groove. With the exception of the congested periphery, the brain was healthy. There was no abscess in the brain. There was slight thrombosis of the sigmoid sinus, the walls being thickened where they lay over the pus in the groove. The lumen of the sinus was still fairly free. The right lung was riddled with abscesses which had become confluent, while the left was only beginning to be affected. It had several wedged shaped infarctions, and the lining membrane of the tubes was highly congested and contained a purulent secretion. There was intense congestion of the intestinal mucous membrane, especially of the upper bowel. In the stomach there was a quantity of muco-purulent material resembling that which was expectorated. There was no indication of tubercle.

Remarks.—The pachy-meningitis and lepto-meningitis radiating from the petrous bone were evidently caused by extension of the infective process from the middle ear through the tegmen tympani. The internal ear seemed free from involvement, though the sheath of the seventh and eighth in the internal meatus was affected with the general lepto-meningitis. There was pus neither in the internal ear nor in the internal auditory meatus. The lungs must have become involved through the blood stream, as there were pulmonary infarctions. It was apparent that he had swallowed his expectoration and probably also purulent secretion from the middle ear passing through the Eustachian tube. This may be regarded as a case of the meningeal pulmonary type of infective sigmoid sinus thrombosis.

En résumé of the symptoms and appearances there were :—

General Symptoms.	1. Infective purulent otitis media of long standing.
	2. Severe pain in head followed by
	3. Rigors which were very frequent, and often succeeded by profuse perspiration.
	4. High temperature.
	5. Rapid pulse.
Pulmonary.	6. Hurried breathing, slight cough at first, with pain in lungs, expectoration prune juice, showing bacteria and pus under microscope. At post-mortem, infective destruction of lungs.
Meningeal.	7. Intolerance of light, irritable temper, fits of screaming, occasional squint, variability of pupillary phenomena.

Toxæmia.

8. The pulse maintained its rapid rate throughout the chloroform narcosis.
9. Sigmoid sinus thrombosis indicated by pain on pressure over the upper thirds of the internal jugular and posterior cervical triangle.

CASE LV. *Infective thrombosis of the right sigmoid sinus, due to chronic otitis media of right side, occasioning infarctions, abscess and gangrene of the lungs. Admitted on the eleventh day of his disease. Duration of the illness eighteen and a half days.*

J. W. (male, aged 26 years), was admitted into Glasgow Royal Infirmary on 29th June, 1888, suffering from otitis media and infective thrombosis of sigmoid sinus, accompanied by inflammation of lung at an early stage, and which developed while under observation into multiple infective abscesses of the lung, followed by gangrene.

History.—*Heredity.*—Patient is one of a family of seven, two of whom died in early childhood. His mother died of phthisis. The rest of the family are healthy. *Personal.*—Patient had been in good health, his only illnesses having been those incidental to childhood. He had a purulent discharge from his right ear during seven years, for which he had been occasionally treated without apparent benefit.

Present Illness.—He dates his present illness from 18th June, eleven days prior to admission, though a week before that he felt out of sorts and was giddy. On this date he was exposed to a cold east wind for some hours, after which he was seized with great pain in the right ear, accompanied by pain in the forehead of such severity as to fairly prostrate him. For forty-eight hours he got no sleep or rest. During this period he was nauseated and vomited once. His mouth and tongue were dry, and he experienced a disagreeable taste and odour. On the seventh day of his illness he had a rigor which lasted half an hour, causing him to shake all over and his teeth to chatter. Two other rigors followed on the ninth day, after which on subsequent days, he experienced sensations of cold without actual shivering. At the termination of the third rigor he became for an hour unconscious, his friends believing it to be a faint. He has had a slight cough, and has lost strength and flesh during his illness.

Condition on Admission.—On entering the hospital on the eleventh day of his illness he appeared to be a well-built man, who had become thin and exhausted. He had a flushed face, and suffused conjunctivæ. He was intelligent, answered questions quickly, though his memory was somewhat defective, especially regarding the incidents of his present illness, which might be accounted for by the confusion which the pain and general disturbance had created. His tongue was coated with a white fur, and his speech was thick. There were complete anorexia and extreme thirst. His temperature was 105°, his pulse quick and irregular, his respirations hurried, his bowels loose, the stools being thin, yellow, and of very offensive

odour. There was frequent micturition, urine being normal. He had a slight cough, just like that of the initial stage of phthisis. At this period his lungs were examined by several physicians, who found no physical evidence in them of anything abnormal. He had no motor or sensory defect in limbs, face, or tongue, though taste and smell were blunted. Reflexes were about normal.

Ear.—Hearing as tested by aerial conduction on the right side was defective, though bone conduction was good. There was a slight discharge of foul-smelling pus from this ear. Of the membrana tympani only a fringe remained at the upper and back part, some connective tissue filling a portion of the upper part, and granulation tissue the lower. There was distinct tenderness on pressure over the upper part of posterior cervical triangle, and along the course of the internal jugular, and also a distinct pain over the right side of the head and back of the ear.

Eye.—The sight was good, the movements of the ocular muscles perfect, and the pupils normal. The ophthalmoscope showed a little congestion of the retinal vessels, but no blurring of the discs. Twitchings of the muscles of the left side occurred very frequently, especially when patient was asleep.

During the first night of his residence in the hospital he complained of severe pain in the chest coming in "stitches" confined to one spot for an hour or so, then changing to another. He could cover each of those painful spots with one or two finger tips. The pain in them was most severe at the onset of each, and gradually died away, though he had an uneasy feeling in the spot for hours after. It may be that the pain would have seemed more prolonged in one place had it not been that his attention was diverted to fresh spots where it was in its initial intensity. There were six such painful spots complained of during the first night, four in one lung, two in the other, and while complaining of them, he repeatedly had a slight "catching cough," which aggravated the pain.

The case was diagnosed as one of infective thrombosis of the right sigmoid sinus, and notwithstanding the absence of physical signs of lung mischief, it was feared that pulmonary infarctions had already occurred.

Operation (30th June, 1888).—The mastoid antrum was opened and found to be a large eroded cavity filled with decomposed inspissated pus and cholesteatomatous matter. The posterior fossa of the skull was opened into at the sigmoid groove, and the sinus was exposed for fully two inches. Between the dura mater and the skull there was a quantity of foul-smelling pus deposited in a layer. The sinus was thrombosed and covered with granulation tissue. The whole of the exposed area was washed out, the granulation tissue removed from the sinus, along with a portion of the thrombus, which had within it a small quantity of pus. The cavity of the sinus was then obliterated, iodoform and boracic acid being introduced, and the parts stuffed with iodoform gauze. The pulse during the chloroform narcosis maintained its rapid rate throughout.

Subsequent Course.—The symptoms were in no way improved by the operation, though there did seem to be a temporary lull. The "stounds"

and "stitches" in the lungs became more frequent and severe, and were accompanied by an expectoration of a slightly yellowish colour composed of pus and swarming with organisms. On the second day after operation, the fifteenth of the attack, coarse crepitation was detected at various points over both lungs, especially at their middle and upper parts, while the lower lobes were free. Two days later coarse crepitation was heard all over both lungs.

On the second day after the operation, the expectoration became prune juice in colour and offensive in odour. Later it was profuse, amounting to nearly twenty ounces per diem, and so foul smelling that patient had to be isolated, as no other patient could remain near him. Diarrhœa had become more marked, the stools yellow and very offensive. The odour from the ear, the breath, and the stools, was of the same kind.

The temperature remained high; rigors, followed by profuse perspiration, were frequent. He rapidly sank from toxæmia and exhaustion, and died at three a.m. on 6th July, eight days from admission, six subsequent to the operation, and on the nineteenth day of his illness.

CHART O.—INFECTIVE SIGMOID SINUS THROMBOSIS.

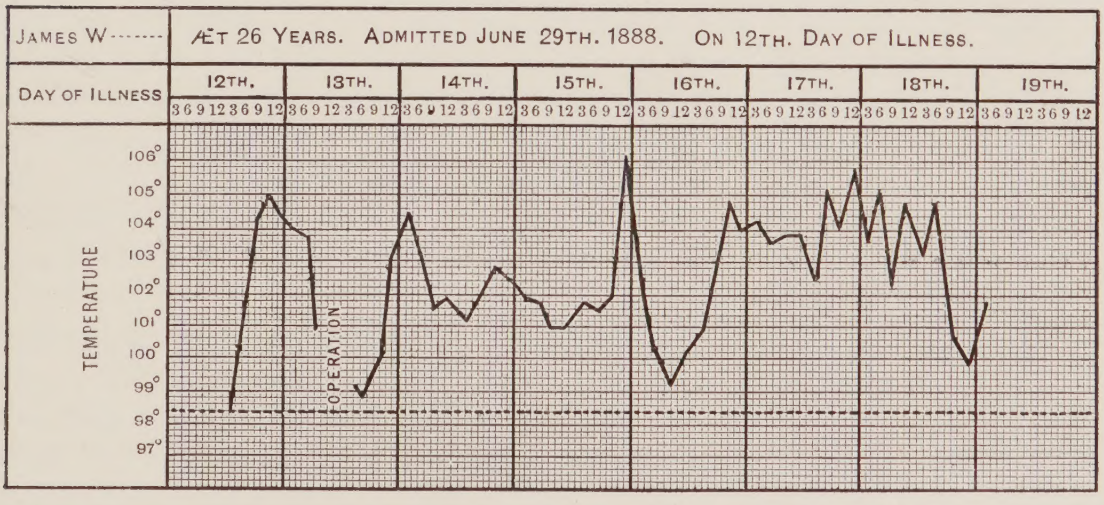


Chart of temperature in case of infective thrombosis of the sigmoid sinus. Toxæmia.—Death.

The temperature chart is given from the date of admission. Drugs were administered during the last three days for the purpose of reducing the temperature. Unfortunately no post-mortem examination was granted.

Remarks.—It was evident without post-mortem confirmation that patient died from infective abscesses and gangrene of the lungs, induced by infective thrombosis of the sigmoid sinus, originating in chronic otitis media. The localized pain indicated the points where the embolisms inducing the infarctions had taken place, and it was interesting to observe that these subjective symptoms were present in the lung before any physical sign of infarction could be detected.

In this case patient complained of a bad taste and odour, which he experienced at a very early period of his attack, and it is possible that this arose from small quantities of infective matter getting into the mouth by

way of the Eustachian tube. If this was the case, the profuse diarrhœa with its offensive odour might have been thus accounted for, though the general systemic affection was quite sufficient to have caused it. In such cases it is also possible for some of the infective matter to be inspired, pneumonia resulting. The whole train of symptoms is here in marked contrast to those of simple cerebral abscess.

CASE LVI. *Infective sigmoid sinus thrombosis with systemic involvement and pulmonary infection; also small abscess in temporo-sphenoidal lobe; both due to chronic otitis media. Death and post-mortem examination. Admitted on twelfth day of illness. Duration of illness fourteen days.*

S. J., male, 46 years, was admitted into the Royal Infirmary, on 6th January, 1887, the twelfth day of his illness, suffering from infective thrombosis of the left sigmoid sinus with systemic invasion, and probably abscess in the left temporo-sphenoidal lobe due to chronic otitis media. He had been seen by author the same evening in consultation with Drs. Garey and Forrest, after which he was removed to the Royal Infirmary.

History.—*Heredity.*—His family history was good. *Personal.*—He was healthy generally. He was a mechanical engineer, and had been regularly at work. For many years he had had a suppurating ear which he was in the habit of cleansing, but which had not been otherwise treated. It had not occasioned him trouble until a few months previously, when he felt pain on the same side of the head, and had in consequence been confined to the house for a few days. When the attack had passed off, he returned to work, and appeared in good health.

Present Illness.—On 25th December, 1886, twelve days prior to admission, he felt out of sorts, thought he had caught cold, and complained of slight pain over the left side of the head. Two days after he vomited, and had a rigor which lasted half an hour, in which his whole body shook. Subsequently the vomiting recurred, and he had rigors with several days interval between each. When seen by his own medical attendant, Dr. Garey, he complained of frontal headache, was drowsy, and gave his answers slowly. His urine was albuminous. His temperature was high.

Condition on Admission.—An hour after his admission to the infirmary, his condition was as follows:—He lay in a drowsy, semi-conscious state, his head being drawn back as in cerebro-spinal meningitis. He could be roused when loudly addressed, and gave monosyllabic answers, a considerable interval after interrogation. He was quite incapable of sustained attention. On several occasions he muttered some words in a low tone of voice, apparently addressed to an imaginary object. He was delirious as he dosed. He could give his name, but could not give an account of his sensations, and did not know where he was.

His face was distinctly flushed, and there was a yellowish tinge in the skin, as also in the conjunctivæ. When asked to put his tongue out, he seemed to have a difficulty in opening his mouth, and therefore it was only

partially protruded, being squeezed between the two rows of teeth. It presented a dry reddish aspect. He opened his eyelids when asked, but owing to the want of sustained attention, closed them after the lapse of ten or twenty seconds. His pupils were equal, rather dilated, and reacted sluggishly to light. There was double optic neuritis. The left membrana tympani was thickened, perforated, and some pus was seen in middle ear. Pressure in upper third of posterior cervical triangle and over course of internal jugular on left side, caused patient to wince and cry out. This was marked. His temperature was 105° F., his pulse was feeble and intermittent, and about 60 per minute. His respirations were hurried, and occasionally laboured. Urine, sp. gr. 1020, contained a considerable quantity of albumin. Occasionally urination was involuntary. There was a general muscular tremor over the body resembling what is seen in the acute exanthemata or in toxæmia when the patient is within a measurable distance of a fatal termination. He had a frequent slight catching cough, accompanied by a fœtid expectoration, which was however only occasionally seen, as it was generally swallowed. All over the posterior aspect of the lungs were moist râles, especially at the right base. His bowels, which had been constipated, were now very loose, the stools having a fœtid odour.

The diagnosis was that of infective thrombosis of sigmoid sinus with infective general systemic involvement. There was also a probability of abscess in the temporo-sphenoidal lobe. It was further highly probable that the lungs had become the seat of infective inflammation. Though patient's condition was very hopeless, it was resolved to open the temporo-sphenoidal lobe.

Operation.—An hour and a half subsequent to his admission the following operation was performed. The mastoid antrum and the middle ear were opened. They contained foul-smelling inspissated pus, which was cleared out. These parts were then rendered as nearly aseptic as possible. Before the temporo-sphenoidal lobe was opened, the foramen for the mastoid vein was exposed behind the mastoid process, and tissues surrounding the foramen on the outside of the vein were minutely examined. They appeared healthy. (This is important in relation to post-mortem.) A trephine opening was made over the squamous portion of temporal, an inch above the external auditory meatus. The bone, the membrane, and the cerebral surface were healthy. The parts exposed were now rendered thoroughly aseptic, and guarded against contamination by having rubbed into them a quantity of powdered boracic acid and iodoform. A hollow needle was inserted into the brain in the direction in which the pus was believed to be situated, downward, forwards, and inwards, towards the roof of the tympanum. It was inserted slowly, and when about an inch of the needle had penetrated the brain, a gush of foul-smelling turbid fluid escaped so suddenly that it was not collected. There was probably about two drachms. This was followed by a stream of pure pus, of a greenish-yellow colour and very fœtid. A portion of this, measuring four drachms, was collected. The passage leading into the abscess cavity was then dilated, a portion of peripheral brain tissue being removed with

this object. A tube was then introduced, through which more inspissated pus escaped. The brain abscess cavity was then washed out with antiseptic solution, several masses of inspissated pus and gangrenous cerebral matter being removed.

Subsequent Course.—After he recovered from the effects of the anæsthetic he declared himself easier, and seemed brighter than previously. Next day, however, the drowsiness returned, there was contraction of the pupil on the affected side, and the temperature was 104° F., about a degree lower than prior to the operation. The muscular tremors over the body generally continued, and towards the end of the first twenty-four hours they increased. The amount of albumin in his urine persisted, his cough was more severe and persistent, the expectoration was foul smelling. He died about forty-eight hours subsequent to the operation.

Post-Mortem Examination.—The head and neck only were permitted to be examined. The brain membranes generally were congested. In exposing the tentorium cerebelli on the left side it was seen to be congested and somewhat softened near its attachment to the petrous bone. The dura mater lining the posterior aspect of the petrous bone, as looked at from the inside, was of a dark yellowish colour, and on both sides of the sigmoid sinus there was a little pus. The position of the sigmoid and of part of the lateral sinus was marked on the cerebellum by a dark brownish tortuous line. On the dura mater being separated from the posterior aspect of the petrous bone, the latter was seen to be covered by a thin layer of pus, which extended down to the sinus and appeared on both its anterior and posterior aspects. The walls of the sigmoid sinus towards the bone were eroded, and had quite disappeared at its anterior and outer knee—the convex bend nearest the mastoid antrum. Here the bone was covered by a quantity of pus and disintegrated thrombus.

The vein which passes through the mastoid foramen was thrombosed, the thrombus occupying the whole of its course in the osseous canal, and just immediately inside of it the bone was covered with pus between the dura and the skull. At its exit from the bone on the outside of the skull, the mastoid was joined by a vein from the upper part of the deeper tissues of the scalp, and this vein, as well as the vein formed by the union of the two, was perfectly healthy. There was no pus in the mastoid foramen.

On making a dissection of the tissues on the outer aspect of the base of the skull, the internal jugular vein was seen to be surrounded by œdema, and at its upper part by an amount of plastic effusion. The vein was not thrombosed, though its walls were thickened. The jugular fossa was, however, blocked with thrombus. The deep tissues behind the insertion of the sterno-mastoid were filled with plastic effusion, an inner and deeper zone presenting purulent infiltration, in the centre of which there was a vein with thickened softened walls, which was traced to the posterior condylar foramen, through which it passed to join the sigmoid sinus. At its junction with the sigmoid sinus the latter contained purulent disintegrated thrombus, which also partly filled the interior of the posterior condylar vein, while on the external surface of the latter pus was found in

connection with that outside the dura in the cerebellar fossa, and which was continuous with that passing through the posterior condylar and anterior condylar foramina to the cervical tissues. The brain was healthy, with the exception of the left temporo-sphenoidal lobe, the surface of which was congested. The abscess cavity was in the inferior and middle convolutions, and extended to within a sixteenth of an inch of the descending horn of the lateral ventricle. What remained of the abscess cavity contained no pus. It was surrounded by a slightly reddish-yellow zone. The surface of the left lobe of the cerebellum was congested; its interior was healthy. There were basal purulent lepto-meningitis, and serous lepto-meningitis of the vertex.

Remarks.—Although the lungs were not permitted to be examined, the evidence all pointed to infective inflammatory action in them, secondary to the putrid thrombosis of the sigmoid sinus. The examination of the exterior aspect of the mastoid foramen during life was made with the view of ascertaining whether there was any inflammatory exudation issuing therefrom. There was none; and the fact that the vein seemed quite healthy, and that there seemingly was a free current of blood flowing through it, pointed to the conclusion that the posterior aspect of the petrous bone was free from inflammatory action. Post-mortem, however, it was seen that the vein running through the mastoid foramen was thrombosed, and that blood could not have flowed through that portion of it. It was also seen that this vein was joined by another from the exterior of the skull, the junction taking place just at the mouth of the foramen. The blood from the exterior vein quite filled the conjoined vein, and thus caused the belief that the mastoid vein was unimplicated. The facts gained from the post-mortem showed that when the posterior aspect of the petrous bone is covered with purulent secretion, the pus does not necessarily show itself on the exterior of the mastoid foramen. Therefore, when there is no purulent secretion or inflammatory exudation at that point, one cannot conclude that there is neither pus on the posterior aspect of the petrous bone nor cerebellar abscess. Further, even if one finds pus issuing from the mastoid foramen, this only indicates that there is pus on the posterior aspect of the petrous bone, and one must not therefore conclude there is abscess in the cerebellum or that there is none in cerebrum. The posterior condylar foramen, being situated at the lowest part of the cerebellar fossa, is the aperture naturally adapted for drainage of this cavity. It must always be borne in mind that both these emissary veins may be absent. The pain experienced on digital pressure in the fossa behind the origin of the sterno-mastoid, which was so sharp as to cause the patient to wince and cry out, was amply explained by the inflammatory zone (tumor albus) round the course of the condylar veins. This inflamed tissue would have formed into abscess had patient lived. The death was here due to secondary pulmonary involvement and toxæmia.

CASE LVII. *Infective thrombosis of left sigmoid sinus and veins leading therefrom, with ulceration of base of temporo-sphenoidal lobe and small abscess between brain and membranes, arising from infective otitis media. General toxæmia. Post-mortem examination. Admitted on fifteenth day of illness. Duration of disease twenty-two days.*

M. L., male, aged 11 years, was admitted into Children's Hospital on 19th January, 1890, on sixteenth day of illness, suffering from thrombosis of left sigmoid sinus, due to infective otitis media.

History.—*Heredity.*—There was a pronounced tubercular hereditary taint, the grandmother and six uncles having died from tuberculosis. A brother, seven months old, suffers from otitis media. *Personal.*—Patient had been in good health, though he had passed through the usual diseases incidental to childhood. When a year old he had left purulent otitis media following an attack of one of the exanthemata. Since then purulent discharge continued to flow till a month ago, when it suddenly ceased. For two years prior to its cessation a little blood was generally mixed with the discharge.

Present Illness.—The meagre facts which could be gleaned were as follows. On 3rd January, 1890, he was exposed to cold and wet, after which he complained of pain in the left ear, which gradually extended over the left side of his head. He frequently vomited during the succeeding days, and on the tenth day of his illness he had a severe rigor. On the fourteenth day it was noted that he had great rigidity of the neck, and when he sat up in bed he supported his head with both hands like a child suffering from occipito-atlantoid disease. He walked with a staggering gait.

Condition on Admission.—On the 20th January, 1890, the seventeenth day of his illness, when he came under personal observation, he was in the following condition. He was an emaciated boy, having an earthy pallor of face though the skin and conjunctiva were yellow and bile-tinged. He wore an anxious, irritable expression. He answered questions intelligently and promptly, although when left undisturbed for a few minutes he was inclined to lie quiet with his eyes shut. It might have been supposed that he was dozing, but any question put to him was answered at once. He complained of pain in the left ear which extended above, behind, and below the auricle. Percussion over the head generally was borne without murmur, but he winced when it was applied to the left temporal region. The sub-mastoid and sub-occipital regions were somewhat hard and swollen, and deep, digital pressure, either there or on the upper two-thirds of the course of the internal jugular caused him to cry out.

Motor Phenomena.—There was slight paresis of the left side of the face, evidently due to involvement of the facial nerve in the temporal bone. With the exception of a few muscular twitchings, which occasionally occurred in the orbicularis palpebrarum, and also erratically in groups of muscles over the body generally, there were no motor phenomena. The reflexes were normal.

Sensation was unimpaired.

Special Senses.—The fundi of the eyes were examined by Dr. Reid, who found them practically normal. The pupils were normal, and patient had complete control over the ocular muscles. The left ear had a number of granulation buds projecting from the upper and back part of the tympanum into the external auditory meatus. The membrane had disappeared. Hearing as tested by ærial conduction was absolutely abolished, and Dr. Barr found that it had also disappeared by bone conduction.

CHART P.—INFECTIVE SINUS THROMBOSIS.

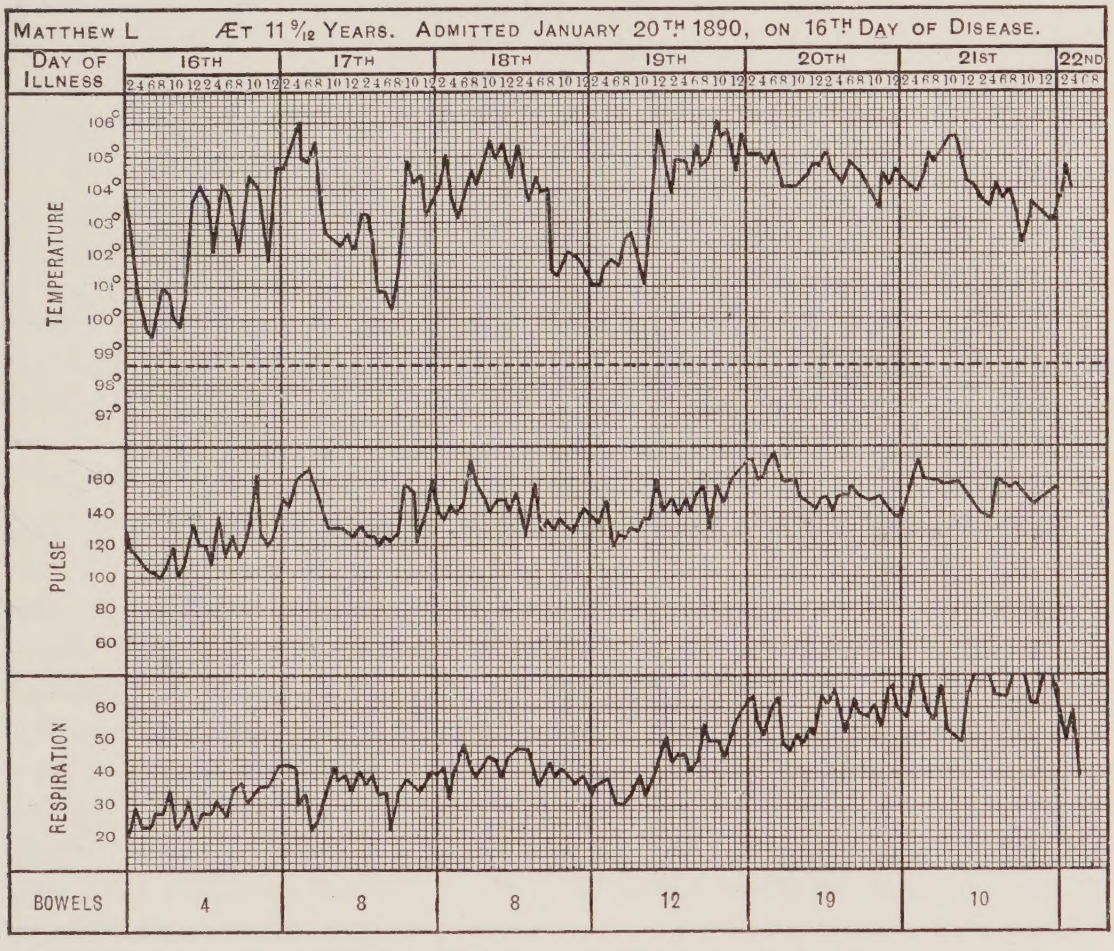


Chart of infective thrombosis of sigmoid sinus—abscess in the temporo-sphenoidal lobe—systemic infection—arising from left otitis media. Death.

Lungs.—Though at this date there were no physical signs of lung involvement, yet patient had occasionally a slight cough suggestive of pulmonary irritation.

He had a very offensive breath, frequently vomited, and was very thirsty, occasionally perspired copiously, had free diarrhœa with thin fœtid stools of a pea-soup character, suggestive of enteric fever. The urine was bile-tinged. The pulse, temperature, and, to an extent, the respirations were all high. The pulse was not only rapid, but very feeble and intermittent.

The Diagnosis was infective thrombosis of the sigmoid sinus, with a degree of meningitis and general toxæmia. Whether there was intracranial abscess was doubtful, but if it exisited its presence was not indicated, the symptoms of the thrombosed sinus being dominant.

On the day succeeding admission he had a rigor, followed by profuse perspiration, and his right lung showed evidences of consolidation over nearly one-third of its extent. Patient was so weak that a partial operation alone was deemed expedient, especially as the system was already involved. It was therefore thought desirable to empty the sigmoid sinus of its contents, if the thrombus was found to be disintegrating, with the view of arresting further systemic invasion.

Operation.—The mastoid antrum was exposed, and masses of granulation tissue, which filled it and the tympanum, were removed, along with fœtid pus. The sigmoid sinus was exposed for about half an inch. There was a layer of fœtid pus in the sigmoid groove. The anterior part of the sinus wall was eroded, and a disintegrating greyish-brown thrombus presented inside. The fluid and pulpy portions of the thrombus were removed, and the sinus at that part obliterated. The tegmen tympani was likewise thinned by erosion. During the time he was under the influence of the anæsthetic the pulse rate never lessened, but was exactly the same as before and after its administration. Subsequent to the operation he appeared to improve for a few hours, after which his weakness became more pronounced, the diarrhœa profuse, the muscular tremors general over the body; and he died from exhaustion on the twenty-second day of his illness. The temperature, pulse, and respiration were taken every hour, and are indicated in the chart annexed.

Post-Mortem Examination.—The more important points are here given:—

Brain.—There was general lepto-meningitis, especially over the left side, where the superficial vessels were much congested. On the dura mater covering the tegmen tympani, and on the anterior surface of the petrous bone, there was an elevated nodule consisting partly of thickened dura and partly of inflammatory plastic exudation. The conical extremity of this nodule projected somewhat into the base of the temporo-sphenoidal lobe. At this point a small subdural abscess was seated containing about a drachm of pus, and associated with cerebral ulceration. The inflammatory exudation forming the conical nodule on the interior of the dura mater was in contact with the pus, the whole being enclosed peripherally by the soldering of the inner membranes to the brain and dura. All the dura in the temporal fossa was much congested, as well as that which covered the posterior aspect of the petrous bone. There was marked thrombosis of the sigmoid sinus, part of its anterior wall having sloughed above and below the obliterated part, the contents of which had previously been removed. The contents of the sinus remaining and situated toward the lateral sinus were of a buff colour, intermingled with greenish-yellow portions—a thrombus in process of disintegration. There was a thin layer of pus between the dura and the skull on either side of the sinus, which spread somewhat over the posterior aspect of the petrous bone, as well as into the cerebellar fossa. There was marked thickening of the sub-occipital and sub-mastoid tissues on the left side, with chains of enlarged glands surrounding thrombosed veins. The thrombi in these veins were likewise disintegrating, and the posterior

condylar contained pus for over an inch of its course beyond the skull. The muscles and other soft structures were matted together by plastic effusion. There was no pus issuing from the mastoid foramen, and the mastoid vein on the outside of the skull was normal. The bases of both lungs were intensely congested, while several distinct infarctions were found in the upper parts. The intestinal mucous membrane was much congested throughout. There was neither tubercular nor enteric intestinal glandular enlargement.

Remarks.—This is a typical case of general toxæmia, arising from infective otitis media occasioning a disintegrating thrombosis of the sigmoid sinus. The general aspect of the patient was distinctly typhoid, and so suggestive was it of ordinary enteric fever as to cause several medical men seriously to raise that question. From the intense inflammation surrounding the disintegrating thrombosis in the veins at the upper part of the posterior cervical triangle, especially in the vicinity of the posterior condylar vein, which contained pus and disintegrating thrombus, an abscess would in all probability have formed, had patient lived. The hearing was absolutely abolished by ærial and bone conduction, yet there was no abscess in the cerebellum and there was a superficial one in the cerebrum.[1] The superficial brain ulceration was in immediate contact with the diseased patch of dura, and the direct continuity of the inflammatory process by contiguity of tissues from the middle ear, through the perforation of the tegmen and various layers of membrane to the brain surface, was well marked.

CASE LVIII. *Summary of two cases of infective thrombosis of sigmoid sinus, with typhoid symptoms, and purulent pulmonary disintegration associated with small abscess in the temporo-sphenoidal lobe.*

There were two cases of sigmoid sinus thrombosis complicated by cerebral abscess of small size seen by the writer in which no operation was performed. A reference only is made to them, as their main features are identical with those given in Case LVII.

The first of the two was in the aural hospital, under Dr. Barr, in 1889. He was about 30 years of age. In this case there were evidences of sinus thrombosis, with lung implication; abscess was supposed to be present though the symptoms were obscured by the dominant thrombosis and the general systemic infection. The symptoms were of a typhoid character, including pea-soup stools, and so strongly resembling those of enteric fever as to cause a physician of great experience to express a doubt as to whether the case was not in reality one of enteric fever, the ear affection being merely incidental. No operation was performed, and the patient shortly after died.

The post-mortem examination revealed thrombosis of the sigmoid sinus, abscess of small size in the temporo-sphenoidal lobe, extensive pulmonary

[1] This contradicts the statement that deafness to bone conduction is pathognomonic of cerebellar abscess.

infarctions and purulent pneumonia. The intestinal mucous membrane was merely injected. There was no intestinal indication of enteric fever.

The second case, seen in private practice in 1890, a male of 28 years, was almost identical; the diarrhœa, rigors, high temperature, the jaundiced skin, and the profuse prune juice expectoration showing the extensive lung involvement. No operation was performed as at this stage the patient was beyond hope of surgical remedy. The patient died twenty-four hours after consultation.

The post-mortem examination showed lesions of a similar character to those described in the above.

Abscess under the Deep Cervical Fascia Resulting from Intracranial Suppuration, or Secondary to Suppuration in the Mastoid.

Case LIX. *Précis of a case of abscess situated in the upper third of the posterior cervical triangle, connected with suppuration in the cerebellar fossa.*

A patient, aged 45 years, was seen by me in consultation in 1886. He had symptoms of basal meningitis for fully a week prior to a swelling appearing in the upper third of the posterior cervical triangle. This was at first very deeply seated, but by and by became more superficial. It was then opened by a surgeon who evacuated several ounces of pus. Two weeks after the opening of the abscess I saw the patient for the first time. He was then in the fourth week of his illness, which was at that date clearly cerebro-spinal meningitis. A discharge of pus still issued from the abscess in the neck. It was fœtid and of a greenish brown colour. The exploring finger found the base of the occipital exposed as far as the condyle, which was bare and somewhat eroded. There was a little discharge of fœtid pus from same ear. The patient, who was in an unconscious state, died twenty-four hours afterwards. No post-mortem was permitted.

The cerebro-spinal meningitis, originating in infective otitis media, was followed by the deep-seated abscess in the posterior cervical triangle which laid bare the occipital condyle, and which was evidently a secondary abscess issuing through the anterior condylar foramen ; or from extradural suppuration issuing through the posterior condylar foramen.

For a case of abscess forming in same position and by extension from cerebellar fossa, see case S. J., No. LVI., 1887.

Abscess in connection with apertures on the internal surface of the mastoid, near the digastric fossa, appear in the upper third of the neck under the sterno-mastoid muscle and the deep cervical fascia, and quickly extend into the upper third of the posterior cervical triangle. They originate

in infective otitis media, the pathogenic processes having extended into the mastoid cells, causing erosion of thin walls near the tip of the mastoid on its inner side. They sometimes assume a large size, and give rise to intense pain, owing partly to the stretching of the tense structures which bound them in and partly to direct pressure on the nerves. The evacuation of these abscesses ought to be undertaken as soon as they are recognized, and it must be accompanied by ablation of the mastoid.

CASE LX. *Précis of a case of abscess under the deep cervical fascia, pointing in the upper third of the posterior cervical triangle connected with perforation of mastoid into digastric fossa.*

A patient, aged 24 years, came under observation in 1893, suffering from a large abscess in the upper part of the left side of the neck, especially prominent in the posterior cervical triangle, but also stretching the sterno-mastoid muscle and curving the neck laterally. He suffered excruciating pain which prevented sleep, and he had the aspect, general appearance, and temperament of one who was suffering from toxæmia. He had had otitis media for many years.

Ten ounces of pus were removed from a very large cavity in the posterior cervical triangle. The base of the skull was bare. During ablation of the mastoid process, which was found to be a shell filled with granulation tissue, an eroded aperture in its inner wall, about quarter of an inch from its extremity, was found, through which the probe passed into the digastric fossa and then into the abscess cavity.

The large cavity rapidly filled with granulation tissue, which soon became cicatrized.

CASES OF SUPPURATION IN THE FRONTAL SINUSES, WITH EXTENSION INTRACRANIALLY OR INTO THE ORBITAL CAVITY.

CASES LXI. AND LXII. *Précis of two cases of suppuration in the frontal sinuses, in each of which there was a perforation into the orbital cavity through a portion of the orbital plate.*

J. M'B., aged 29 years, a fisherman, was sent to author by Dr. Freeland Fergus with an abscess which pointed above the upper eyelid near the nose, which was found to be connected with an erosion in the orbital plate of the frontal opening into the right frontal sinus. He had had an offensive purulent discharge from both nostrils for some months previously, and suffered from dull aching sensations, which he described as a weight over the brow, and occasionally from dull heachaches over vertex. He had afterwards an acute inflammatory attack, preceded by a shivering and intense pain "in

the eye," which subsided when the eyelids closed from the swelling. This swelling subsided after the abscess burst.

The frontal sinuses were freely opened, when the mucous membrane was found to have disappeared, and a mass of granulation tissue filled with infective purulent secretion occupied the sinuses. This was removed, the infundibula rendered patent, the necrosed erosion on roof of right orbit removed, and the whole aperture treated so as to produce firm occlusion by connective tissue. He was afterwards seen absolutely cured.

Six weeks after this man presented himself for the first time, his sister (M. M'B.), a girl of 16 years, came to be examined for a precisely similar

FIG. 56.—INFECTIVE INVASION OF THE FRONTAL SINUS WITH INTRA-ORBITAL ABSCESS.

The characteristic appearance of intra-orbital abscess arising from pyogenic extension from the frontal sinuses. The sinus is seen near eyebrow.

affection, and as her appearance was typical of such cases, the face was photographed. (See Figure 56.) In both the pus from the frontal sinuses was examined by cultivation and found to contain the same pyogenic micro-organisms, the streptococcus pyogenes and the staphylococcus salivarius pyogenes (Biondi). The girl had probably been infected from the brother.

CASE LXIII. *Précis of case of infective intracranial invasion from pyogenic infection in the frontal sinuses.*

In a case which occurred in the wards of Mr. Millar, surgeon to the Royal Infirmary, Edinburgh, to whom the author is indebted for the information, the patient, a girl of 18 years, who had a tubercular family history,

was admitted with suppuration in the frontal sinuses of over eighteen months' duration. She had persistent pain over the left frontal sinus, accompanied by dull headache of frontal region and top of the head. There was a discharge from the nose of pus which was thick and yellow. She complained of pain on the left side of chest, but there were no physical signs of intra-thoracic disease. The temperature was subnormal. The left frontal sinus was opened and syringed out through the nose. This relieved her for a time, but further progress of the infection was evident in the state of the frontal periostitis, high temperature with remissions, dulness of the lungs at their bases posteriorly, enlargement of the spleen, and other evidences of general systemic infection from which she died. At the post-mortem examination the right frontal sinus was found full of pus, the left, the one which had been operated on, was empty. There was pus between the skull in the frontal region and the dura mater, and over the same region there was pus on the brain surface extending to the longitudinal fissure. The longitudinal sinus was full of pus. There were no thrombi in the lateral sinuses. No osseous erosion between the frontal sinuses and the brain. In the right lung there was recent pleurisy. At the base of the left lung there was a wedge-shaped area of infarction, surrounded by yellow softening. A second similar area was found higher up in same lung. The spleen was large, soft, and congested.

It is not evident in this case from which frontal sinus the infective disease spread inwards to the brain.

Infective Invasion of the Intracranial Structures without Visible Wound.

Case LXIV. *Précis of case of intracranial extradural suppuration with pachy-meningitis, exhibiting "Pott's puffy tumour," originating in infective bruising of the scalp and deeper tissues the result of a blow.*

J. R., aged 45 years, received from the shaft of a cart a severe blow on the left side of vertex, about an inch from the middle line. He was subsequently able to work for fully a week without feeling anything wrong except slight pain over the part. Subsequently he felt feverish, the pain which was of a dull character increased, and was accompanied by occasional sharp stounds over the vertex. He also had great headache and prostration. When examined there was a distinct puffy tumour over the seat of former injury. This swelling, patient declares, came on three weeks after the accident, and after it had formed he had some relief to the pain. The primary swelling from the bruising had subsided some weeks before the puffy swelling appeared. On incision the skull was found bare, a small quantity of semi-purulent exudation bathing the bone. The diploic tissue of the bone was filled with granulation tissue, which could be traced in small portions

penetrating the bone, both through the external and almost through the internal table of the skull, which was dark in colour. Between the internal plate of the skull and the dura there was a considerable layer of freshly-formed granulation tissue bathed in purulent exudation. His symptoms quickly disappeared after the operation.

In this case either the bruised-softened brain had been infected at the time of the accident through apertures too small for recognition (vaccination scratches), or the streptococci, which were found in abundance in the dural and diploic granulation tissue, had already been in the interstices of the skin of the scalp—the glandular prolongations or involutions about the hair bulbs. The ingress of the organisms once accomplished, they developed apace in the midst of the bruised and softened tissue.

CHAPTER V.

TREATMENT.

Prophylactic.—Abscess in the brain, infective thrombosis of the intracranial sinuses, and lepto-meningitis originate in primary infective foci. The first step in prophylaxis is to prevent the occurrence of such foci, the second to eradicate them when present.

Rendering the parts aseptic in scalp wounds and compound fractures of the skull.—In scalp wounds and compound fractures of the skull, the hair ought first to be closely shaven, the scalp thoroughly cleansed with soap and water aided by a soft nail brush. The parts should then be dried, rubbed with turpentine to remove unctuous matter, dried a second time, and soaked for a few minutes in methylated spirits, after which an aqueous solution of carbolic acid (1-40) is applied for at least an hour before the wound is dressed. In preparing the skin for any operation, the same course is pursued, and whenever possible, after adopting the preceding measures, the parts are soaked in a carbolized water dressing for 48 hours prior to the operation.

Should the edges of the wound or the surfaces of the flap be ingrained with dirt which cannot be otherwise removed, these parts ought to be pared with a sharp scalpel, so as to leave a fresh aseptic surface.

When the skull is bruised, indented, and ingrained with dirt without fracture, great care is necessary in order to remove the whole of the infective matter. This may best be done by slicing off the outer layer of bruised and infiltrated bone by means of a sharp chisel. The periosteum ought to be closely scrutinized, as dirt is occasionally deposited between it and the bone, where it is apt to be concealed.

If fracture exists, the edges of the fractured bone ought to be inspected, and if impregnated with foreign matter, they ought to be refreshed with a chisel. A trench may be formed, by removal of bone from both edges of the fracture, to permit of easy access for the application of cleansing processes to the diploë and inner table, or, if necessary, a disc of bone may be elevated to ensure the complete removal of pathogenic organisms. The bone, after being rendered aseptic, may be reimplanted.

Wounds of dura mater and brain.—When the dura has been penetrated, and the inner membranes, or the brain itself, inoculated with infective products, the dura ought to be freely opened, in order to expose for inspection the inner membranes, as well as the brain; also to facilitate the removal of foreign matter, and prevent the ultimate extension of deleterious products. When, in addition to the puncture of the brain and its membranes, the cerebral substance has been bruised, it is very difficult in the midst of disintegrating brain tissue and extravasated blood to ensure the removal of all extraneous matter. When doubt on this point exists, it is preferable to leave the wound open—exposed to the influence of iodoform gauze,—first to permit of the free escape of inflammatory products, should they unfortunately form; and secondly to secure the direct application of antiseptics.

All sources of suppuration or infective matter in the orbital, nasal, buccal, oral, and pharyngeal cavities, and in the maxillary and frontal sinuses ought to be removed, or, when this is not practicable, the discharges ought to be rendered and maintained aseptic. This is sometimes difficult without extensive operation in lesions of the nasal fossa or its annexa, but as long as an infective source remains it is an element of danger and a menace to the patient. Cario-necrosis of the skull should be similarly dealt with. The frontal and maxillary sinuses, when in a state of persistent suppuration, may require trephining to afford free vent to the pus, and for the purpose of removing from the walls infected granulation tissue and eroded bone. The proximity of the frontal sinuses to the brain render infective processes in them very hazardous, especially when accompanied by erosion of their posterior wall. When the frontal sinuses are involved, both ought to be exposed. The neglect of this precaution in one case, reported to author, led to a fatal issue from suppurative lepto-meningitis, originating in the opposite frontal sinus from that which was exposed.

Infective pustules about the nose, face, lips, or neck, require immediate excision, and the ligation of the main veins conveying the blood from the diseased parts. In infective wounds of the orbital cavity, rather than permit the formation of infective thrombosis of the cavernous sinus, the serious question of extirpation of the eyeball, and clearing out the contents of the cavity may arise.

Acute purulent otitis media seldom occasions intracranial complications, though diphtheria or other infective disease of the mouth, tonsils, and pharynx, may extend into the middle ear by way of the Eustachian tube, and by further extension set up lepto-meningitis or infective sinus thrombosis. When such occurs, the invasion is generally so rapid that the intracranial involvement occurs before opportunity is given for radical intervention, though direct and prompt application of antiseptics to the middle ear and mastoid antrum and cells aids in preventing the further extension.

As chronic purulent otitis media and the extension of inflammatory processes to the mastoid antrum and cells is the primary focus which leads most often to intracranial inflammatory lesions, the eradication of the *otitis media* must be regarded as the most potent factor in the prophylaxis of inflammatory cerebral lesions.

The treatment of middle ear disease—too frequently neglected.—Where the tympanic cavity has become the seat of chronic suppuration, with ulceration of the mucous membrane extending into the antrum and mastoid cells, it becomes a standing menace to the safety of the patient. The disease progresses insidiously, and one cannot be certain as to where and when it may end. A person might as well have a charge of dynamite in the mastoid antrum and cells, as one cannot know the moment when accidental circumstances may arise which may cause the infective matter to become widely disseminated all over the cerebro-spinal system. It is no part of the plan of this work to enter into the general treatment of aural disease, which is fully discussed in the many excellent treatises on diseases of the ear, to which the reader is referred. Chronic otorrhœa is, however, much too lightly regarded, and is frequently considered as a mere inconvenience, instead of a menace to life. It is true that many affected with chronic purulent otitis media pass through a long life without suffering obtrusively from it, and may die from causes neither directly nor indirectly associated with it. On the other hand,

the disease is one which advances insidiously, often without pain, until very extensive destruction of the middle ear and the mastoid antrum and cells, with thinning of their osseous walls, and serious invasion of the meninges, the bloodvessels, and the brain itself occurs. The mere localized suppuration, continuing for many years, is a source of weakness tending to waxy or amyloid degeneration of the tissues. As long as the middle ear and its recesses are the seat of pus secretion, they are liable to become a focus for pathogenic organisms which may find their way into the general circulation, or by extension invade the meninges and brain in the manner described in the pathological and clinical sections. From this very small focus, the brain and cerebro-spinal meninges may become affected. One who has a chronic purulent otitis media is liable to have, with very little warning, a most serious, or even a fatal illness. Besides those who succumb to such diseases recognized during life, others do so from disease directly or indirectly the result of chronic otitis media and its extensions, but which are registered under the heading of its prominent sequelæ pneumonia, pleurisy, meningitis, etc. It is a fact that the records of most aural hospitals show that their patients are all confined to the earlier decades of life. This may be variously interpreted, but it is probable many people die from such sequelæ before they reach forty years.

Under these circumstances, all persistent purulent discharges from the middle ear ought to be regarded seriously, and the medical attendant ought not to rest satisfied until the discharge is cured. If this cannot be accomplished after sufficient trial by the ordinary means at the disposal of the aurist, applied through the external auditory meatus, or Eustachian tube, then recourse ought to be had to operative treatment, directed, first to arresting encroachments of the disease toward the cerebral and cerebellar fossæ; secondly, to opening the various recesses of the mastoid process and removing therefrom diseased membrane, granulation masses, cholesteatomatous debris and bone erosions contained therein ; thirdly, to affording a free outlet to discharges which are otherwise pent up; and fourthly, to filling up these parts with fresh fibrous tissue, or to forming a permanent opening to the middle ear through the antrum.

It must be borne in mind that cases do occur where *caries sicca* (tubercular) takes place in the mastoid without rupture of

the membrana tympani, others in which purulent secretion finds its exit by the Eustachian tube, the membrana tympani remaining intact. When a patient suffering from either of these becomes suddenly affected with an intracranial lesion, the proper cause and sequence of the brain lesion is apt to be overlooked.

Indications for opening the mastoid antrum.—The chief indications for exposing the mastoid antrum and cells are the following:—

1. Repeated inflammations of the mastoid antrum and cells, accompanied by swelling over the mastoid process, or where there is a fistulous opening in the bone discharging pus.

2. Acute inflammations of the mastoid antrum and cells with retention of pus within them.

3. When there are initial symptoms of intracranial involvement associated with chronic purulent otorrhœa, the exposure of the mastoid antrum and cells ought to be immediately undertaken, and the whole diseased parts in the middle ear cleared out.

4. Persistent chronic otorrhœas, not principally due to the condition of the tympanum or Eustachian tube, and which have long resisted approved treatment or which are deemed by an aurist otherwise incurable, even though there be no abscess, œdema, or inflammatory indication over the mastoid. If, in addition, the otorrhœa (even suppose it be odourless) be found to be infected with virulent pathogenic organisms, or if it be highly offensive, mixed with osseous debris or cholesteatomatous masses, these offer an additional incentive to opening the mastoid antrum, and clearing away the masses of granulation tissue from the middle ear, the mastoid antrum and cells. The most serious intracranial mischief—infective lepto-meningitis, sigmoid sinus thrombosis, and cerebral abscess—arising from otitis media, are often present without marked mastoid swelling.

With the opinions of Schwartze upon the indications for opening the mastoid antrum and cells, the author thoroughly agrees.

Preparation of the patient for operation upon the mastoid antrum.—The patient ought to be in as good a state of health as his condition is likely to permit. The middle ear ought to be rendered as nearly aseptic as possible. The hair for a considerable area behind and above the mastoid ought to be shaven, the whole area of operation, and the surrounding parts, cleansed with soap and water, then with turpentine, and finally with methylated

[1] Gruber, *op. cit.*, p. 432 ; also see *Archiv für Ohrenheilkunde*, Bd. xiv., S. 202.

spirits. After this has been done, a water dressing consisting of lint saturated in a watery solution of carbolic acid (1 to 40) and covered with gutta percha tissue ought to be applied over the parts. If time permit, this water dressing ought to be maintained for over 24 hours prior to operation.

The *instruments required* are a scalpel, a couple of sharp retractors, a periosteal elevator, a bur or other perforator, a specially-constructed gouge for raising thin superficial slices of bone, a few hand instruments for exploring and widening the various apertures, others for removing granulation tissue and osseous particles from the middle ear with its annexa, and a Eustachian catheter. These, with a few artery or pressure forceps, are all the instruments required. Ligatures ought to be at hand, though they are seldom needed. Minute portions of sponges, or, as the author prefers, shreds of aseptic gauze are required for the constant cleansing of the parts between each application of the instruments. A watery solution of carbolic acid (1 to 40) for cleansing hands and instruments, and a weaker solution (1 to 100) for washing out the mastoid antrum and middle ear, are necessary. A mixture of iodoform and boracic acid (1 to 4) is used for filling the interstices of the bone, along with iodoform gauze. A good lamp with a reflector—the ordinary laryngeal mirror answers well, or, better still, the electric light—for illuminating the recesses of the mastoid antrum and the tegmen tympani, etc. Besides the anæsthetist, two assistants are necessary—to steady the head, to retract the soft parts, and to sponge the wound.

The operation of opening the mastoid antrum.—The mastoid antrum is the key to the position in all operations whose aim is to expose the mastoid cells. Once the antrum has been opened, the further enlargement of the aperture in the direction of the mastoid cells may be undertaken with safety, and through the passage between the antrum and middle ear, or by its enlargement, the latter may be thoroughly cleansed. The main point in the operation is to expose the antrum, and, while doing so, to avoid—first, opening the sigmoid groove and injuring its enclosed sinus; secondly, encroaching upon the Fallopian canal, and destroying the facial nerve; thirdly, invading the middle fossa of the skull; and fourthly, injuring the semicircular canals.

The patient having been anæsthetized and placed on his side with the affected mastoid uppermost, the external ear being held

well forward by one assistant, while another steadies the head, the surgeon delimits the mastoid process, and, when operating on the patient's right side, places the forefinger of his left hand upon the posterior zygomatic root, which projects behind and above the ear, and his thumb upon the tip of the mastoid process, (when operating on the patient's left side, the position of the operator's thumb and finger will be reversed, if the operator stands on the left side of the patient,) and makes with a scalpel a perpendicular linear incision, about a quarter of an inch behind the posterior border of the external osseous meatus, extending from a third of an inch from the tip of the mastoid to the posterior root of the zygoma. This incision penetrates at once the periosteum, which is then elevated forward along with the soft tissues, so as to separate the whole of the post-auricular attachments and fully expose the posterior aspect of the external auditory meatus. The post-auricular artery, the bleeding from which has been meanwhile controlled by the pressure of the thumb against the extremity of the mastoid, is now caught by pressure forceps and turned aside, as are likewise any smaller vessels which may have been cut. The reflected tissues, along with the auricle, are held aside by a sharp retractor. The tissues to the posterior side of the linear incision are thus left adhering to the bone.

Three points to be noted in the conformation of the bone.—The surgeon then notes the three following points in the conformation of the bone. First, the position of the supra-meatal triangle—a triangle formed by the posterior root of the zygoma above, the upper and posterior segment of the osseous external meatus below, and an imaginary perpendicular line uniting these two, extending from the most posterior portion of the external osseous meatus to the zygomatic root (see Figs. 57 and 58). Within this triangle, and touching its base, the opening into the mastoid antrum may be made with safety. Second, the degree of obliquity of the posterior osseous wall of the external auditory meatus, as, when this wall is directed more obliquely from behind forward, the mastoid antrum is situated slightly more anteriorly than when the osseous wall of the meatus is directed more transversely from without inwards. Third, the depth of the inner wall of the tympanic cavity, from the level of the skull at the osseous portion of the external auditory meatus, which may be determined by introducing a probe through the external ear till it comes gently into contact with the inner wall of the tympanum, the membrane

having previously been perforated by pathological processes, and then marking on it the limit of the outer aspect of the osseous meatus. If the middle ear lie deep, the mastoid antrum, which is more superficial, may be expected to be relatively deeply seated.

These three points being noted, the operation may proceed. The opening is to be made at the base of—but within—the triangle (Fig. 57), the perforation being directed inwards, and a little forwards, so

FIG. 57.—SUPRA-MEATAL TRIANGLE.

Schema of squamo-mastoid portion of the temporal, showing the supra-meatal triangle in relation to the sigmoid groove and facial nerve.

as to expose the antrum. As long as the excavation is made in this direction, and within the triangle, the sigmoid groove cannot be injured. In brachycephalic skulls the middle fossa of the skull may lie very close to the roof of the external ear, and where the interspace is very limited the roof of the external osseous meatus may require to be encroached on during the perforation. In many cases the antrum projects behind the base line, and below the level of the triangle, but its recesses in these directions can be reached more safely after the antrum has once been opened from within the triangle.

The safest instrument for the purpose of exposing the antrum is the rotatory bur (see Fig. 60), propelled by a surgical engine, driven by hand or water power,[1] the hand-piece of which is held in the hand like a pen. The size of the bur to be used will depend upon the dimensions of the triangle. A small one will be used in the child, and a large one in a wide triangle. This instrument applied lightly to the bone removes osseous flakes, leaving a smooth, almost polished surface, upon which any dark objects, such as are presented by the interstices of the mastoid antrum or any of the adjoining cells, are readily recognized previous to being opened. When one of these small dark spaces is exposed in the bone, a searcher (see Fig. 60) or suitably constructed probe ought to be introduced into it, and the dimensions of the aperture determined. If it communicates with the antrum, the searcher will enter a wide cell, leading into the middle ear. Very frequently in the pathological conditions spoken of, when the wall of the antrum is penetrated, an oozing of pus occurs, or a bud of granulation tissue protrudes. This is by no means diagnostic of the antrum, however, as the same may occur in certain pathological conditions of the sigmoid sinus, and of the dura mater over the tegmen tympani. If the aperture communicates with the antrum, a small bur is applied to its margins until the whole of the external wall of the antrum is removed, the searcher being repeatedly used during the process to ascertain the antral dimensions, and to follow its main irregularities.

The depth at which the mastoid antrum lies from the surface varies, according not only to the anatomical peculiarities, but also to the superimposed pathological changes. As a rule, in children the antrum is fairly superficial, even when the discharge has been very chronic; but during adolescence, and in after life, the persistence of caries or cario-necrosis is very often accompanied by hypertrophic sclerosis, which augments the thickness of the bone outside of the antrum. It also encroaches upon the limits of the antrum itself. Occasionally the antrum is absolutely obliterated. The depth at which the antrum may be situated from the surface varies from one-eighth to three-quarters of an inch, or even more. When it is not exposed after penetrating to a depth of half an inch, great care must be exercised in avoiding the facial nerve. This may be best done by keeping

[1] The electro-motor can be used, but the supply of electricity is often uncertain—unless when used from storage cells—and requires more attention.

close to the floor of the middle fossa, and nearer the posterior border of the opening towards the postero-superior angle of the supra-meatal triangle. On two occasions (the patients curiously enough coming from the same village, and being near neighbours and having brachycephalic skulls), the antrum was situated more internally, and somewhat above the level of the outer limit of the floor of the middle fossa, the downward projection of which had to be rounded in order to reach the antrum. It is possible in some of the reported cases in which the mastoid antrum could not be found during operation, that it was situated in this manner.

When the antrum has been fully exposed, its interior is cleansed of all pus, cholesteatomatous masses, and granulation tissue. When this has been done a scrutiny of the interior of the antrum is made.

Points to be noted when the antrum is exposed.—Determine first, the position of the passage between the antrum and the middle ear. Secondly, the position of the facial canal, which generally traverses the inner half of the floor of the antral passage obliquely from without inwards, as it passes into the inner wall and roof of the tympanum above the foramen ovale. It is often marked on the floor of the antral passage by a cylindrical ridge of smoother, denser bone than the surrounding cancellated osseous tissue. When, however, the mastoid antrum is deeply seated, from superimposed masses of sclerosed bone, then the texture of the facial canal cannot be distinguished from that of the eburnated tissue surrounding it. In such cases the antrum and the middle ear are apt to be limited in size, and the position of the facial canal is somewhat obscured, and its contents may therefore be endangered during operation. In these the antrum ought to be approached along the upper part of its external wall, so as to avoid the nerve. An assistant ought to be placed in a position to observe any facial twitchings and to announce their occurrence. These twitchings of the facial occur when the rotatory bur encroaches on the contents of the canal. Another danger to which the facial nerve is exposed arises when a granulation mass, protruding through an extensive erosion in the canal, encloses the facial nerve, the surgeon in clearing the granulation tissue from the floor of the antrum is apt to injure the nerve. Before removing granulation masses from the floor of the antral passage, the granulation buds ought to be touched with a probe or other

suitable instrument, when, if they enclose the nerve, facial twitching will result. When all these precautions have been taken the nerve is rarely injured, though the possibility of its occurrence must be considered as one of the risks of the operation. Thirdly, the state of the tegmen antri ought to be carefully examined for osseous erosions. When granulations project from the tegmen they ought to be closely examined, lest they be protrusions from the dura mater. If they be found to protrude through the tegmen in this way, that is evidence of an external, though it may be a localized, pachy-meningitis.

Exposure of the mastoid cells.—If the mastoid cells behind and below the antrum are involved, they ought to be exposed, their external wall obliterated, and their noxious contents removed. It is safest to do this by working from the exposed antrum downwards and backwards, always examining the exposed cavities and apertures with a searcher or probe before the removal of bone. In working over the mastoid region the sigmoid groove ought to be borne in mind. It frequently happens that granulations, springing from the dura lining the cerebellar fossa, and covering the sigmoid sinus, protrude through the sigmoid groove, and project into one of the mastoid cells. A knowledge of this fact may prevent mistakes being made, as such buds of granulation tissue look at first sight as if they grew only from the mastoid cells. When these granulations project in this way from the cerebellar dura, there is a localized pachy-meningitis, and probably a more or less thrombosed sigmoid sinus. After all the disease is removed from the mastoid antrum and cells, their walls ought to be rubbed down with the bur, so that no partitions are left, the whole being thrown into one cavity with bevelled edges and smooth polished walls.

Opening the tympanic attic.—If the state of the middle ear, previously ascertained by auriscopic examination, renders it desirable to open into the tympanic attic, this may be done by means of a fine bur applied to the junction of the roof with the outer wall of the antral passage. Neither the floor of the passage nor its inner wall must be encroached on, lest the facial nerve or the semicircular canal be injured. Sufficient bone ought to be removed to freely expose the attic. The tegmen covering the tympanic cavity can then be examined by means of a beam of electric light. The antral passage is in many instances sufficiently wide, without further enlargement, to permit of the

thorough cleansing of the attic and the remainder of the tympanic cavity.

Removal of the ossicles.—If the malleus and incus are eroded, their mucous membrane is destroyed, and they are covered with granulation tissue, and their ligaments softened. They likewise can be easily removed through the antral passage by means of minute hooks and ear scoops. To leave either of these ossicles in the middle ear covered with granulation tissue, and in a state of erosion, is to court further extension of disease.

Effect upon the function of the ear.—The stapes ought to be left *in situ* unless there be some adequate reason for its removal. When it, or the membrane covering the fenestra, is left, hearing by that ear generally is sufficiently good for ordinary conversational purposes, and sometimes it is so well that patients have declared they have heard quite as acutely with the ear from which both incus and malleus have been removed as with the other which was in a normal condition. By the application of finer tests, however, a defect in hearing was detected in the ear operated on as compared with its healthy neighbour. On several occasions the stapes was likewise removed in the midst of granulation masses, and hearing by those ears, though defective, was still retained to loud conversation. One patient heard a watch at one inch from that ear, while the same watch was heard at twenty inches from his normal ear. It is probable that the membrane covering the fenestra ovalis was retained.

In one instance, where an opening was kept patent between the external surface of the mastoid and the middle ear, hearing was much more acute when the posterior aperture was left patent than in the opposite ear.

Destruction of chorda tympani, and its results.—It is almost impossible to avoid destroying the chorda tympani—if the disease has not previously destroyed it—while removing the malleus and incus in the midst of granulation masses. This destruction results in the loss of taste in the anterior two thirds of the tongue on the affected side. This defect, when unilateral, is very seldom recognized by patients unless attention has been specially directed to it.

Means of facilitating the closure of the gap in the mastoid antrum.—If there be any special reason for rapidity of filling up the gap left by ablation of the antrum, this may be greatly facilitated by removing, after opening the antrum, a considerable portion of the

osseous wall of the external auditory meatus at the junction of its roof with its posterior wall. The softer tissues of the external ear tend more quickly to fill up the gap to which they are brought by contraction and by granulation tissue once the osseous barrier has been removed. Some authorities have recently advised filling the gap by uniting the edges of the superficial wound with stitches. This would not be advisable unless the aperture in the mastoid was previously filled with cicatricial tissue, so as to prevent re-accumulation of putrid matter.

Choice of instruments for opening the mastoid antrum.—Instead of using the bur throughout, the operation may at certain stages be facilitated, especially if the bone be soft, by using a gouge for the removal of some of the superficial layers of the bone. In doing so the gouge ought to be placed on the bone at the base of the triangle and made to cut forward towards its apex. The rounded head of the instrument lies in the hollow of the hand, while the index finger of the same hand guards its cutting edge. It then acts as a plane raising thin shavings of bone. The posterior bevel toward the cutting edge of the instrument greatly favours this action (see Fig. 60).

Objections to the use of the chisel and mallet.—Most surgeons use the chisel, driven by a mallet, for opening the mastoid antrum. No doubt it is often successfully exposed in this way, and in lack of a better instrument the chisel may be used. Its use, however, for opening the mastoid antrum is objectionable. First, because in operating upon a restricted area, surrounded by, and full of apertures, some containing delicate structures, the wounding of which may occasion serious and even fatal results, the antrum being situated at a variable depth from the surface, the intervening osseous tissue being of various consistency, it is a source of danger to use a chisel driven by a mallet, which may be suddenly impelled into the interior of one of those spaces or into the cerebellum, brain, or sigmoid.

Such accidents have occurred. During attempts to open the mastoid antrum the sigmoid sinus has frequently been accidentally opened by the chisel, several of the cases surviving, though a number have died. In four unpublished cases which have been communicated to the author, four surgeons, in their several endeavours to open the mastoid antrum, have accidentally driven the chisel into the sigmoid sinus. In each of these there was profuse hæmorrhage, causing an abandonment of the operation,

the parts being plugged to arrest bleeding. Three of these four patients ultimately died. In two the surgeons believed that the death was not hastened by the accident, and from the meningeal complications previously existing it is probable that a fatal issue would have occurred in any case. In one the amount of hæmorrhage within a few seconds was very great. The patient died in forty-eight hours from extensive cerebro-spinal meningitis, and a hæmorrhage of considerable size was found in the subdural space in the same cerebellar fossa. The fourth case recovered.

On one occasion a young surgeon, in endeavouring to open the antrum with a chisel found on giving it an impact with the hammer that the instrument suddenly entered a large cavity, from which pus issued. At post-mortem examination it was seen that the chisel had penetrated the sigmoid groove, pushed aside the sinus, which was fortunately thrombosed, and entered the cerebellum, from which, on withdrawal of the instrument, pus flowed.

Secondly, the repeated blows with the hammer impart considerable vibration to the mastoid and adjoining structures, and when the tegmen tympani or the sigmoid groove is eroded and thinned the concussion may cause these thinned and friable structures to give way, or cause a rupture of the granulations covering them, and thus permit inoculation of the fresh wounds in the membranes, or in the brain itself. Pus within the brain or cerebellum, receiving through the skull a succession of violent vibrations or shocks, could easily be scattered, or a large abscess near the ventricles or meninges might, by the violent impacts conveyed to it, burst into the ventricles or meninges, thus producing serious if not fatal results. On one occasion (in a case reported to the author by the surgeon in charge), while the chisel and mallet were being vigorously applied to open a mastoid antrum, the patient's breathing was suddenly arrested; the relations of a large cerebellar abscess which was present being probably disturbed by the communicated vibrations. There was no laryngeal obstruction.

Thirdly, the aperture left by the chisel is rough and rugged compared with the polished surface and bevelled edges obtained with the bur, which greatly facilitate the after-dressing and treatment.

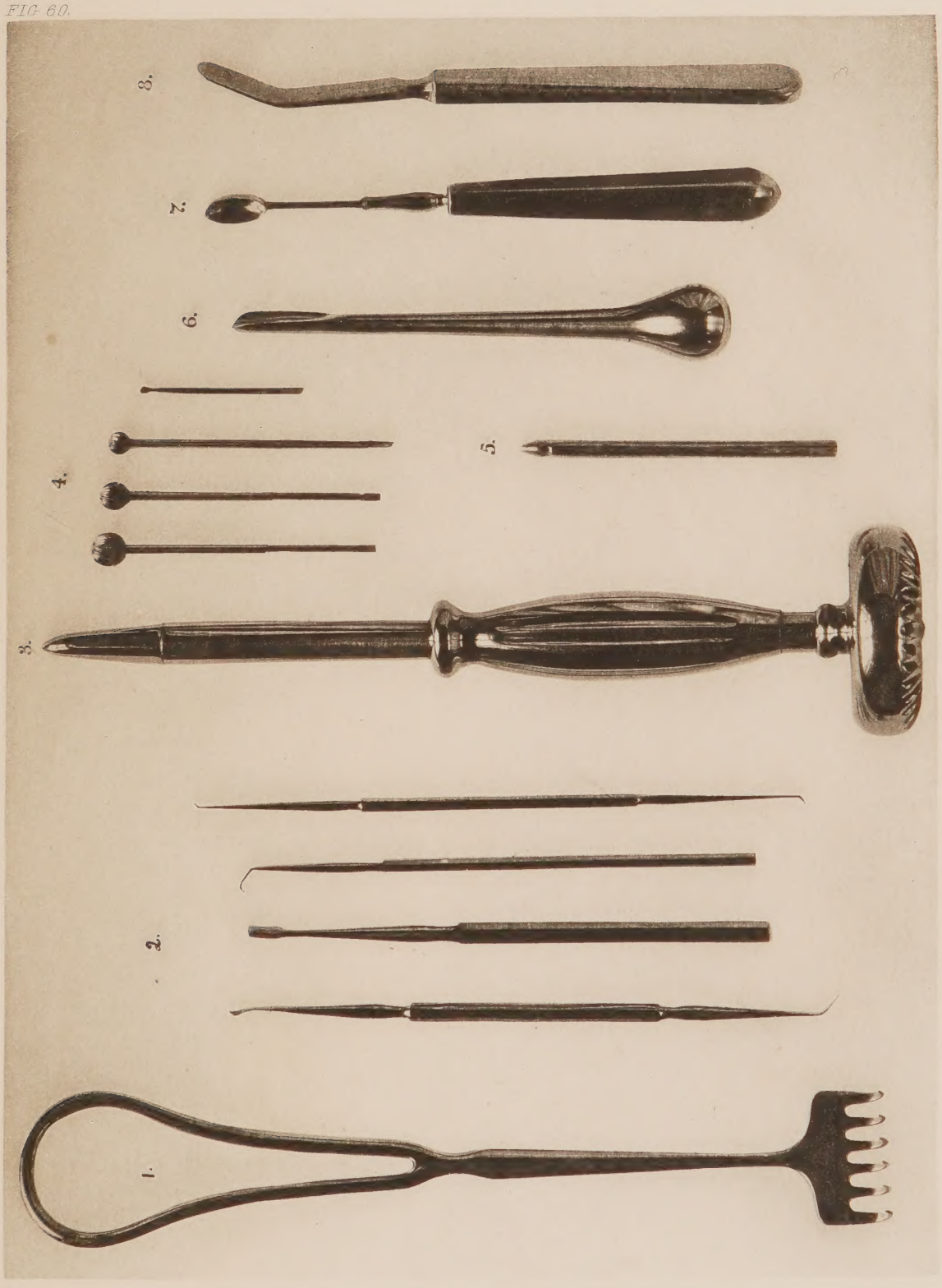
A Russian instrument for perforating bone is of service in boring an aperture through the bone into the antrum. It works easily, and the bone debris which it pares off is gathered into the

FIG. 60.—INSTRUMENTS.

1. Retractor. 2. Set—Searcher and scoop, chisel, single and double ossicle hooks. 3. Bone-perforator. 4. Set of Rotatory burs. 5. Cerebral exploratory cannula. 6. Mastoid gouge. 7. Volkmann's spoon. 8. Periosteal elevator.

To face page 304.

FIG 60.



handle instead of being scattered about. Were it to be much used, various sized cutting points would require to be constructed for it. On several occasions it has been employed by the author in exposing the antrum in children, and though not equal to the bur, and requiring to be used with greater care, especially in soft bone, it yet answers the purpose very well (see Fig. 60).

The use of the trephine for opening the mastoid is, like that of the chisel, attended with danger, and is not advised.

Caution with regard to injecting fluid from the antrum into middle ear.—In the ordinary position in which patient's head is placed for operation on the mastoid antrum—lying on its side with the affected ear uppermost—if fluid be injected through the antral passage into the middle ear, instead of issuing by the external auditory meatus, which is thus above the stream and at right angles to it, it will pass for the most part through the Eustachian tube, which is more in line with the stream issuing from the antrum. When the injected fluid reaches the pharynx, while patient is anæsthetized, it is apt to enter the larynx, and, if in large quantity, may cause temporary respiratory obstruction, and convey infective matter into the lungs. This is obviated by allowing the patient's head to hang over the table before injecting; the fluid will then flow through the nose, from which it may be removed. It may also be prevented by introducing the Eustachian catheter, and reversing the stream, which ensures the cleansing of the Eustachian tube, the middle ear, and the antrum. Before this is done, it is preferable to inject air through the tube to ensure its patency, and by occluding the external ear at the same time the fluid in the middle ear and antrum will be expelled by the antral opening.

In testing the communication between the middle ear and the antrum, by means of an aseptic solution, the auricle ought not to be pulled firmly forward, otherwise the external auditory meatus may be occluded, and thus prevent the escape of fluid from the ear.

After the ossicles have been removed, a strip of gauze may be passed from the external ear through the middle ear into the mastoid antrum, removing granulation tissue, and cleansing the route. When the stapes is to be conserved care must be taken to prevent it being injured—both in manipulations in the middle ear and also in the passage of tubes or dressings from the antrum to the middle ear.

Dressing the antrum and middle ear.—When the antrum, middle ear, and external auditory meatus have been thoroughly cleansed and dried, they are loosely filled with iodoform and boracic acid (1 to 4), and lightly stuffed with iodoform gauze, a strip of which is also inserted into the external auditory meatus as far as the middle ear, and stuffed so as to co-apt the soft tissues of the external auditory canal to the posterior osseous wall, or, where that has been removed, to the exposed antrum. The iodoform is of use not only in restraining the growth of the pathogenic organisms and rendering their products innocuous, but the powder prevents the close adhesion of the gauze to the tissues, and therefore facilitates the removal of the latter at subsequent dressings. The above is supposed to last for a week or more without redressing. If the parts have to be dressed daily, boracic acid would be used in the subsequent dressings.

Objects aimed at during wound healing.—All infective matter having been removed from the mastoid antrum and cells, the subsequent treatment aims at preventing the possibility of any reaccumulation of such. All the mastoid cells that are continuous with the antrum being obliterated, and the whole of the external wall of the antrum being removed, one of two terminations may be aimed at; either to fill the antrum and tympanic attic, when it is affected, with granulations which ultimately become converted into firm avascular connective tissue, presenting an efficient barrier to the further invasion of the tegmen antri and that portion which covers the tympanic attic on the one hand, and to the invasion of the sigmoid sinus by the commonest route on the other; or to establish a permanent passage from the middle ear through the antrum to the surface of the mastoid from which any discharge forming in the middle ear or petrous portion of the temporal may have free escape while the tegmen and sigmoid sinus are guarded by the new vesture of connective tissue, covered by a non-secreting layer of epithelium. When there is no evidence of petrous or internal ear disease the former is chosen; when there is such, the latter is preferable, as it is also when there is any possibility of subsequent meningitis. In each case the wound must be made to heal from the bottom, and the stuffing of iodoform gauze ought to be placed like a wedge with its base on the outer level of the wound.

When a permanent opening is desired, the passage from the middle ear to the external antral surface must be "papered" with

epithelium, part of which extends from the mucous membrane of the middle ear, and part from the skin until they coalesce, the former sometimes extending more rapidly than the latter. In furtherance of this end, a narrow strip of gauze ought at each dressing to be passed through the antral passage to the middle ear. This is the method usually adopted in the author's practice. The frequency of the dressing depends on the nature of the case and the amount of discharge. The author has in fifty-four cases, uncomplicated by intracranial lesions, opened the mastoid antrum, removed the mastoid cells, and cleansed the middle ear.

THE TREATMENT OF INFECTIVE SIGMOID SINUS THROMBOSIS is directed toward preventing the dissemination of, and destroying the infective products. The primary source of the lesion must first be eradicated. When this is due to chronic middle ear disease, the mastoid antrum and cells are dealt with as already described. Then the seat of the infective thrombus is fully exposed, and if possible any infected tissue lying between the primary source of the disease and the sinus, is taken away. In doing this, the osseous covering of the sigmoid sinus ought to be freely removed, so as to allow sufficient access for manipulation, antiseptic ablution, and the introduction of antiseptic gauze or other form of dressing. When infective thrombosis has formed, every hour is of importance if any attempt is to be made to arrest the progress of the disease. The patient's strength ought to be maintained by the administration of milk or other nutritious liquid, or semi-solid predigested foods.

To expose the sigmoid sinus in the sigmoid groove an incision is made over the prominence of the mastoid, about a quarter of an inch anterior to its posterior margin, extending from the posterior root of the zygoma to near the tip of the mastoid process. The soft tissues along with the periosteum are reflected in one piece by means of a periosteal elevator. At the upper aspect of the incision the suture at the parieto-squamo-mastoid junction ought to be exposed. This is the anterior extremity of the parieto-mastoid suture (see Fig. 58) —not the asterion, which lies at the posterior extremity of the parieto-mastoid, from half an inch to an inch and a half behind. The parieto-squamo-mastoid junction is well marked and easily found. A line drawn from this point to the tip of the mastoid process gives the course of the sigmoid sinus (see Fig. 58). In the adult this line marks the middle

of the sinus, but occasionally it strikes its posterior border. On the left side this line more frequently corresponds to the anterior border of the sinus. An osseous opening, with its posterior margin touching this line, and within the parallels of the roof and floor of the external osseous meatus, will expose the part of the sigmoid sinus most often affected. Within these parallels, but especially in line with the upper, the most superficial portion of the sigmoid sinus is found usually a quarter of an inch, though it may be only about one-twelfth of an inch from the surface. It is thus much more superficial than the antrum. About the level of the floor of the external auditory meatus, the sinus passes deeply into the skull toward the base. The asterion corresponds to the union of the lateral with the sigmoid sinus. The parieto-squamo-mastoid junction corresponds to the union of the sigmoid and superior petrosal sinuses and also to the posterior upper ridge of the petrous bone as it abuts upon the mastoid. Slightly below this point is situated the knee of the sigmoid sinus, which, having its convexity forward, is the most anterior part of the sinus, and the part which is most frequently affected with pathogenic processes spreading from the antrum.

In the adult a vertical line drawn half an inch behind the posterior osseous wall of the external auditory meatus, and between the levels of its roof and its floor, will, in the majority of instances, indicate the position of the middle of the diameter of the anterior convexity of the sigmoid sinus.

The opening into the sigmoid groove ought, by preference, to be made with a spherical bur, which more safely exposes the sinus in its irregular groove than any other instrument. After making the first breach in the wall of the groove, it may be used to enlarge the opening to any extent desired.

The sigmoid sinus is, however, usually exposed subsequently to the opening of the mastoid antrum, in which case it is preferable to open the sinus from its anterior aspect, and in doing so, to obliterate the bridge of bone between the antrum and the sinus, which so frequently contains the channels through which the pathogenic organisms reach the sinus. The anterior knee of the sinus is situated from one-eighth to a quarter of an inch behind the base line of the supra-meatal triangle. The posterior wall of the mastoid antrum being exposed, an opening is made in the bone behind that point for half an inch horizontally, which, in the majority of cases, will open the greater part of

FIG. 58.—SURFACE GUIDES FOR SIGMOID SINUS.

Three artificial lines drawn upon the skull, indicating the following:—(1) The short vertical line from the posterior border of the external auditory meatus to the posterior root of the zygoma marks the base of the supra-meatal triangle. (2) The second vertical line, extending from parieto-squamo-mastoid junction to tip of mastoid; the upper two-thirds of its length indicates the position of the sigmoid sinus. (3) The oblique line, passing from asterion to upper limit of external auditory meatus, indicates in its posterior two-thirds the sigmoid sinus from its commencement to its knee.

To face page 308.

the diameter of the sinus, after which the sigmoid groove may be opened above and below that point as the disease indicates.

Frequently, in opening the sigmoid groove, granulation masses protrude from the dura mater covering the sinus, and often along with these there is an oozing of pus. If it be considered advisable to open the sigmoid sinus, and turn out its disintegrating contents, then fully a vertical inch of the sinus ought to be exposed before opening it, in order to facilitate the operation and the subsequent measures necessary for its obliteration. Occasionally, the wall of the sinus next the bone is ulcerated, and the sigmoid groove being opened, the contents of the sinus become exposed to view, when the disintegrating matter may be removed by the aid of a small spoon, or gently washed out—the former is the safer. Any space existing between the sigmoid groove and the sinus ought likewise to be cleared out and rendered aseptic. Granulation tissue protruding from the dura mater ought first to be carefully examined with the probe, lest it surround a sinus communicating with the cerebellum, and leading into an abscess. If such a sinus exist, it requires to be opened, and the cerebellar abscess dealt with. If there be no sinus, the granulation tissue ought to be removed, as it is apt to harbour infective matter in its sinuosities.

The obliteration of the sinus.—After the interior of the sinus has been rendered as far as possible aseptic, a mixture of iodoform and boracic acid, 1 to 4, is thickly powdered over its interior.

Its external walls which were previously split are folded inwards, so as to obliterate the lumen of the vessel. A quantity of iodoform and boracic acid powder, sufficient to fill the interior of the cavity existing between the obliterated sinus and the bone, is introduced, care being taken in doing so not to exert so much pressure as to strip the sinus or surrounding dura from the skull. As in these cases an infective wound is being dealt with, the external portions of it are best stuffed with iodoform gauze, so as to permit free drainage, and healing by granulation tissue. By obliterating the sigmoid sinus over about an inch of its extent, the influx of blood from the lateral and superior petrosal sinuses is arrested, and the efflux through the mastoid vein is likewise cut off.

How to occlude the sinus in the case of hæmorrhage.—In the event of hæmorrhage occurring on removal of the thrombosed

mass on the distal side of the sinus, the steps just detailed for the obliteration of the sinus would suffice to effectually arrest it. Were hæmorrhage occurring from a wound in a non-thrombosed sigmoid sinus, it would be best dealt with by detaching with a periosteal elevator a portion of the external wall of the sinus from its osseous groove, then indenting the loosened wall into the lumen of the vessel, where it is maintained by catgut, or iodoform and boracic acid powder placed between the bone and the vessel, and supported externally by gauze stuffing.

If the wound be a mere slit in the sinus, pressure exercised upon the spot for a few minutes often serves to occlude it.

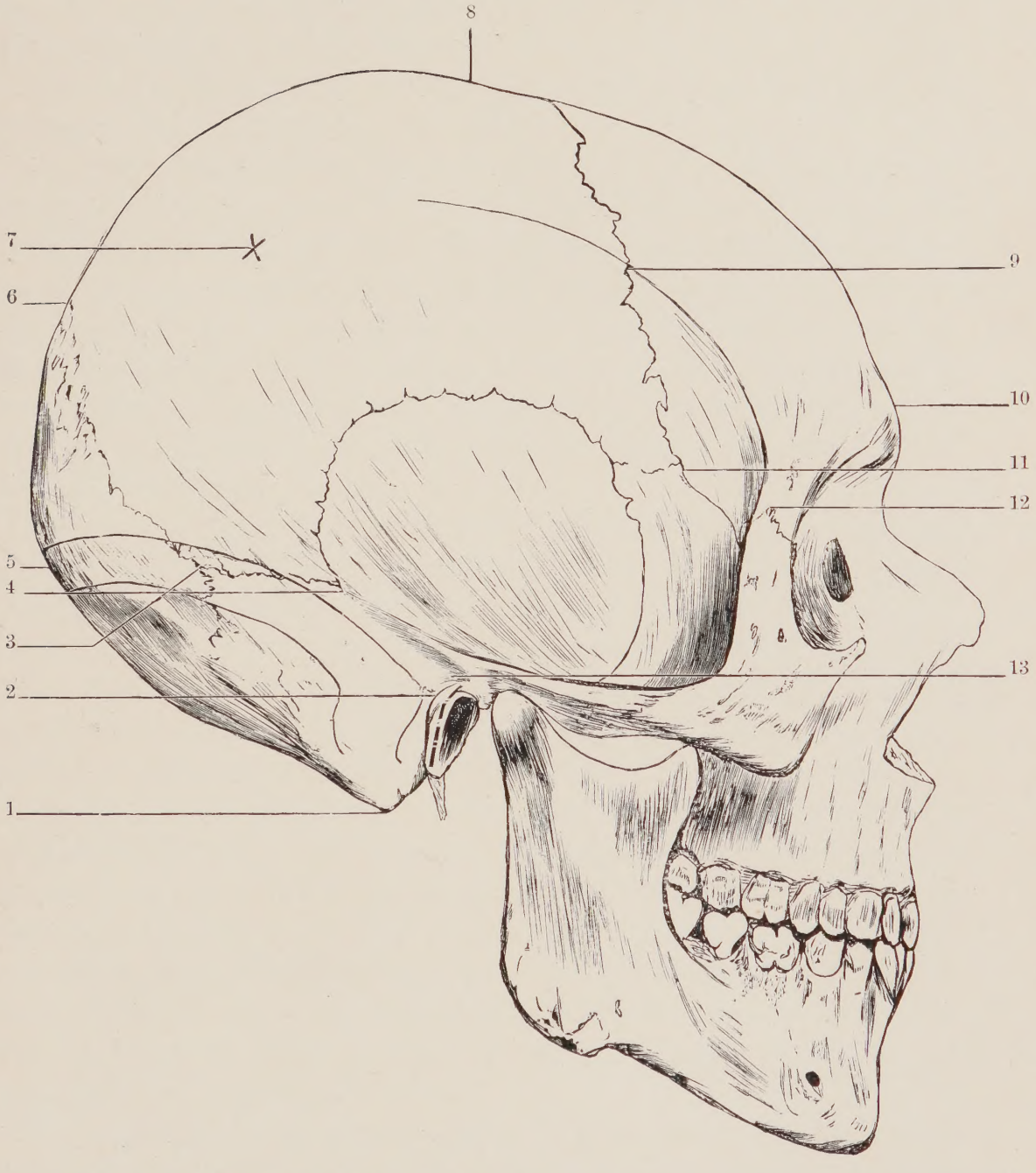
The admission of air into the sinuses.—In manipulating the contents of the sinuses, especially when removing the disintegrating thrombus from the side nearest the jugular bulb, care is necessary against the admission of air, more especially if aseptic washings be employed. It is best avoided by removing the disintegrating clot on that side by means of the small, sharp spoon. The mechanism of the jugular bulb prevents the aspiration of the sinus, and air will probably not be admitted unless the jugular bulb be encroached on.

Any attempt to ligature a large sinus is attended with difficulty and danger, and is generally futile. An aperture in the bone, sufficient to expose the breadth of the sinus, would be required, and in passing the needle care would require to be observed in order to make it go through the dura mater beyond the sinus, instead of transfixing the latter. The soft brain tissue on which the sinus rests, and the large meningeal veins which pass into the sinus, are apt to be penetrated or ruptured during the attempt. After the ligature has surrounded the sinus the walls are so wide and resisting, that they are approximated with difficulty, and are apt to be torn during the process.

Ligation of the internal jugular vein for thrombosis of sigmoid sinus.—In cases of infective thrombosis of the sigmoid sinus, ligation of the internal jugular vein in two places, and dividing it between them, has been proposed by Mr. Horsley, and performed by Mr. Ballance and Mr. Horsley, in addition to the removal of thrombi from the sinus. In certain cases this procedure is useful, and ought to be performed.

Where the infective thrombus in the sigmoid sinus has undergone such extensive disintegration as is unlikely to be reached by obliteration of the upper two-thirds of the sigmoid sinus, the liga-

FIG. 59.—SURFACE MARKINGS ON SKULL.



1. Tip of mastoid. 2. Roof of osseous meatus. 3. Asterion. 4. Parieto-squamo-mastoid junction. 5. Inion. 6. Lambda. 7. Parietal eminence. 8. Bregma. 9. Stephanion. 10. Glabella. 11. Pterion. 12. External angular process of frontal. 13. Supra-meatal triangle.

To face page 310.

tion of the internal jugular is indicated. When in a thrombosed internal jugular, giving the sensation of a hard cord-like structure, its upper part becomes soft from disintegration of the thrombus, and when this is followed by descending disintegration ligation of the vessel below this point is in most cases necessary. When one can foresee in the outset of the case that the vein requires to be ligatured, it had better be done as the first step, before clearing out the sigmoid sinus, as greater freedom may then be obtained while performing the latter.

It must not, however, be forgotten that the internal jugular, though the main channel, is not the only one between the sigmoid sinus and the lungs. The posterior and anterior condylar veins, and the occipital sinus, though much smaller channels, are yet large enough to permit the passage of minute particles of disintegrating clot to the lungs by the numerous venous routes entering the subclavian, and consequently the mere ligation of the internal jugular would not suffice to prevent dissemination of infective particles.

When the upper two-thirds of the sigmoid sinus is obliterated, not only the blood from the lateral sinus, but that from the superior petrosal as well, is shut off from flowing through the sigmoid, while the exit by the mastoid vein is likewise occluded, and the blood from the inferior petrosal sinus, entering the jugular bulb, would flow directly into the jugular vein, away from the sinus, provided the internal jugular be patent. When, however, it is ligatured the flow is reversed, passing from the inferior petrosal into the lower part of the sigmoid, and through the smaller channels, the anterior and posterior condyloid veins, and the occipital sinus. In thus reversing the stream the debris from the lower part of the sigmoid may be swept onwards into the lungs. In the majority of instances the obliteration of the upper two-thirds of the sigmoid sinus is all that is necessary for the prevention of systemic infection.

It must also be recollected that in some cases the connective tissue in the sub-mastoid and sub-occipital regions is infiltrated with infective products, and even the tissues within and surrounding the carotid sheath are occasionally involved in infective serous extravasations. In such not only would the ligation of the internal jugular be more difficult, but the beneficial results would be doubtful, and the wound made would probably not be aseptic.

When there is clear evidence of general infective invasion, and the lungs are implicated, the internal administration of antiseptics may be of benefit. The pulmonary conditions must be treated on general principles.

Anæsthetics in inflammatory cerebral affections.—In acute inflammatory cerebral affections anæsthetics ought to be administered with great care, as the brain is often already œdematous, and a prolonged or very profound anæsthesia increases the œdema. The anæsthetic ought to be chloroform, never ether. The period of struggle ought to be avoided by giving the chloroform very gradually. A very small dose of morphia, $\frac{1}{8}$th gr. suppository, facilitates the anæsthesia, but as the effects of even small doses of morphia occasionally last longer than is desirable, and may obscure the mental phenomena exhibited, its omission is preferable. After the skull and dura have been opened, the patient as a rule experiences very little pain, even if conscious, and, therefore, the anæsthetic may be discontinued. Children, however, are best kept under its influence until the operation is completed. Anæsthetics are especially dangerous in the case of cerebellar abscess, where the brain is often œdematous, and the ventricles greatly distended with serous fluid. A very slight increase in the serous fluid may cause arrest of the respirations.

Infective meningitis, extradural abscess, and abscess of brain in temporo-sphenoidal lobe.—When the patient has presented symptoms of infective meningitis, extradural abscess or abscess of the brain, the mastoid antrum and the tympanic attic having been fully exposed, the tympanum ought to be carefully scrutinized, granulation tissue growing from it removed, and any fistulous openings leading intracranially explored, and their position relative to the exterior of the cranium noted.

Extradural abscess above the tegmen.—By enlarging the opening in the tegmen, extradural abscess may be thoroughly evacuated. The extent of bone removed must be determined by individual circumstances, but it ought to be sufficient to prevent re-accumulation of fluid between the dura and the bone. No pockets or crevices are to be left, and all granulation tissue should be removed. It is safe to open the tegmen freely in an outward direction from the seat of the perforation. Before injecting fluid —if such be deemed necessary—between the skull and the dura ascertain the limits of the extradural space, and whether there be an opening through the dura into the inner membranes or

the brain. If no such opening exist, then the extradural cavity may be washed out with safety, and dressed with iodoform and boracic acid powder and iodoform gauze.

Caution about injection of fluid through apertures in the tegmen.—The forcible injection of fluid, through a small aperture in the tegmen, without having previously explored the dural covering, may result in stripping the softened dura mater from the bone, over a portion of the squamous and also over a portion of the base of skull, and disseminating the infective particles over these areas. Also when an aperture exists in the dura, fluid thus injected may be forced through into the membranes and brain, likewise carrying with it and disseminating inflammatory products. Such injected fluid may be retained, doing harm by its physical presence.

In numerous instances of infective lepto-meningitis the free opening of the membranes, the clearing away of the infective intra- and extra-dural pus and rendering the cavity aseptic have resulted in complete cure of the patient.

Ulceration of brain at base of temporo-sphenoidal lobe.—When pus issues through an aperture in the dura mater from the intra-dural structures, after the tegmen has been opened sufficiently and the extradural surface cleansed, the dura is opened freely. If the pus be found gathered in the arachnoid or pial meshes, or if it issues from ulceration of the brain surface, it may be thoroughly dealt with from the opening in the tegmen. The inner membranes ought to be opened, and the cerebral surface washed with a stream of antiseptic solution, and dusted with antiseptic powder. No drainage tube is required if the openings through the tegmen and dura have been sufficiently free.

TEMPORO-SPHENOIDAL ABSCESS.—When pus is found to issue through the dura above the tegmen from a cerebral abscess in the temporo-sphenoidal lobe, the abscess may be evacuated by enlarging the aperture in the tegmen, and also extending the aperture outwards through the squamous portion of the temporal. Such an opening into the cerebrum suffices for temporary purposes, but though it always ought to be made in order to eradicate the source of the infection, it is not safe to trust to it alone, as in many cerebral abscesses there are sloughs of brain tissue which cannot be easily removed in this way, but require a larger opening in the skull for their evacuation. If these sloughs be allowed to remain in the brain, they retain infective

matter, which maintains the irritation, and may give rise to fresh abscess formation.

Position of opening through the external aspect of the skull.—In abscess of the temporo-sphenoidal lobe, it has been proposed to make the opening through the skull at various points. It ought to be made as near to the seat of disease as possible. In the majority of cases the abscess in the temporal lobe originates in the tissues directly above the tegmen tympani or antri, and therefore an opening in the squamous portion of the temporal above the tegmen will be the nearest point to the cerebral abscess.

When the mastoid antrum has already been opened, the incision in the soft parts made to expose the antrum is extended upwards for a couple of inches above the posterior zygomatic root. The soft tissues including the periosteum are reflected by the periosteal elevator, and a disc of bone half an inch in diameter is removed. The centre pin of the trephine is placed at a point in line with the posterior osseous wall of the external auditory meatus, and three quarters of an inch above the posterior root of the zygoma.

Mode of operating.—As the squamous portion of the temporal is one of the thinnest parts of the skull, the trephine guard at first ought to be placed about an eighth of an inch from the cutting edge of the teeth of the instrument. The trephine disc ought to be placed as evenly upon the bone as possible, but it must be borne in mind that irregularities exist on the cranial aspect of the bone, the inner table being not always parallel to the outer, which may permit the trephine to penetrate the whole thickness of the skull at one part sooner than at another. A narrow bladed searcher (Fig. 60) is frequently introduced into the groove made by the trephine with two objects—first, to clear away the sawdust; second, to ascertain the depth to which the trephine has penetrated. This ought to be done all round the disc, as the skull may have been penetrated at one part, though not at another. When one portion of the bone has been penetrated before the rest, the remainder is removed by a few half turns of the disc confined to that area. The periosteal elevator is now used as a lever, being introduced between the button and the skull, the latter being used as a fulcrum. When the button of bone which has been thus elevated is intended for re-implantation, it is at once placed inside of an aseptic sponge, and kept in warm aseptic solution until it is required.

When the dura is exposed, any purulent secretion which may exist extradurally is removed, and the colour and appearances of the dura are noted. If it be normal, and especially in a young subject, the course of the larger pia matral vessels may be discerned through the dura from their dark-blue colour. In large abscess of the brain, the pressure is often sufficient to empty the superficial bloodvessels, which lie collapsed, and which will therefore not appear through the dura. Before opening the dura, it is well to cover the exposed osseous surface and its cut edge with the iodoform and boracic acid powder, rubbing it into the cut osseous surface, in order to protect these parts from contamination by the infective pus about to be withdrawn. In incising the dura, commence toward the centre of the opening, when, if any of the pia matral vessels are injured, they can be much more easily secured than if they were cut at the margin of the osseous aperture, beyond which they may retract and bleed into the subarachnoid and subdural space. The first incision in the dura ought to be in line with the larger pia matral vessels. In dividing the dura, do not penetrate its whole thickness at once, but pick up its various layers until an opening has been made, when a blunt flat director may be introduced between the dura and the soft membranes, and the remainder of the dural incision completed. A cross cut may likewise be made, to give freer access to the brain and soft membranes.

Pus or plastic effusion may be present in the arachnoid meshes, when there has been localized suppurative lepto-meningitis, quite independently of abscess in the brain. When the cerebral abscess is large, there will probably be bulging of the brain into the trephine opening, and, if it be situated near the aperture in the skull, there will probably be an absence of cerebral pulsations. A small abscess may, however, exist at a deeper level without any diminution of the cerebral pulsations. When there has been lepto-meningitis, accompanied by pachy-meningitis, the membranes may all be fused one to another and to the surface of the brain, which will then not impart a sensation of fluctuation even suppose a large abscess is superficial. Occasionally fluctuation may be detected in the brain when the abscess is large, and not far from the surface.

With the object of exploring for pus, and removing it when found, either of three instruments may be used—a cannula armed with a trocar, a hollow needle, or a pair of sinus forceps. The

instrument ought to be inserted in an inward, downward, and slightly forward direction, so as to impinge, if it went far enough, against the cranial aspect of the tegmen tympani. While the instrument is being inserted, a *slight* to and fro lateral movement ought to be imparted to its point, with the view of ascertaining whether it has entered a cavity, inside which its extremity can move without resistance. When the sinus forceps are used the blades are several times gently expanded as they are introduced.

If the hollow needle without a stylet, such as some of those used with the aspirator, be introduced, it will cut the brain substance and become occluded, and if the pressure inside the abscess be small, the occluding brain substance may not be forced out of the hollow needle, and consequently any pus which may be in the brain may be prevented from escaping. It is possible that the abscess cavity may be missed if attention be not directed to this point.[1] The introduction of a trocar and cannula will obviate this difficulty, but while being inserted it is liable to transfix a small abscess, without revealing the pus, unless the trocar be removed, once for each quarter of an inch of brain tissue penetrated. The use of a cerebral exploratory cannula, such as is here figured, serves the purpose better than other instruments of kind mentioned. While its extremity is occluded, it has several apertures at the sides through which the pus gains an entrance into the tube (see Fig. 60).

The escape of pus and removal of sloughs.—When the pus is found, its rate of flow from the cannula varies according to the amount of intracranial pressure, and when the abscess is becoming emptied the flow will be influenced by the respirations, increasing during expiration. Along with the pus, molecular debris and minute sloughs of brain tissue may be extruded through the cannula, but the larger sloughs, which are so frequently present in brain abscess, cannot escape in this way, they require a large opening for their removal. For this purpose, a quantity of brain tissue, existing between the abscess and the surface of the brain, may require removal, or the peripheral brain tissue may be turned aside. While the cannula is retained in the abscess cavity as a guide, the cerebral tissue may be removed by means of a Volkmann's spoon, or it may preferably be turned aside by introducing a pair of dressing forceps closed and opening them *in situ*.

[1] Two such cases are recorded, see Clinical Reports.

Frequently, when the forceps have thus been expanded, one or more of the sloughs flow between their blades and can at once be removed. Once the opening in the cerebral tissue has been sufficient, the sloughs are often extruded by the intracranial pressure, and are carried slowly to the surface of the brain. If the sloughs be too large to be thus extruded, they may be gently detached from the abscess wall, and assisted out by the forceps and the sharp spoon, it being necessary to discriminate carefully between the slough and the healthy brain tissue. It is of importance to remove these sloughs thoroughly, as if they remain they are apt to set up fresh irritation and abscess in their periphery by the infective matter which impregnates them, and which is contained in their interstices. The retention of such sloughs is one of the most fruitful sources of reproduction of abscess after evacuating the primary one, and is also a cause of delayed healing. It is true that were they rendered aseptic they could be absorbed, but this would be difficult to secure when they arise from an infected source. The leaving of cerebral sloughs is as dangerous as not eradicating the primary source of the infection or the channels by which it spreads to the brain.

Washing the abscess cavity to render it aseptic.—After removal of the sloughs, the cavity is washed out with a boracic or weak carbolized watery solution (1-100). In order to do this a second cannula is introduced by the side of that first inserted, the calibre of the second being at least a half greater than that of the former. A stream of antiseptic fluid is caused to flow gently through the cannula with the finer calibre, and allowed to find an exit by the larger tube. It need scarcely be added that the stream is to be gently introduced, and any resistance met with must not be overcome by forcing the fluid through, but by ascertaining that the tubes are not occluded by debris from the abscess cavity, and this applies especially to the exit tube. Fluid introduced with considerable force into the abscess cavity would burst it and disintegrate the brain tissue, and might inflict irreparable damage. The surgeon must be satisfied that the fluid is going into the abscess cavity and that it returns by the exit cannula, otherwise he must desist from injecting.

On one occasion a surgeon, believing he was washing out an abscess, injected several ounces of fluid into the subarachnoid space, the most of which was retained, causing a widely dilated

pupil on the same side, and a dissemination of the infective particles.

After the stream of antiseptic fluid returns clear, before the removal of the cannulæ, the head of the patient ought to be turned so as to allow the fluid to gravitate out. One ought to bear in mind that the fluid introduced may cause dispersion of the pus and dissemination of the infection, unless within a firmly formed capsule. In the absence of proper flushing arrangements instead of washing out the abscess cavity the introduction of the iodoform and boracic mixture is advisable.

Double opening into abscess cavity.—Where the abscess is seated near the floor of the middle fossa or has a direct communication with it, the opening in the tegmen ought to be enlarged so as to gain access to the abscess cavity from below, if this has not already been done. The larger cannula inserted at this point serves to remove the fluid injected through the finer cannula introduced by way of the opening in the squamous, and more thorough washing of the abscess cavity is thereby secured. This aperture in the tegmen and membranes serves afterwards for drainage purposes, for which it is admirably situated, being at the lowest level of the abscess cavity when the head is raised on a pillow. When this method is adopted the Eustachian tube and the external ear are both shut off by filling the middle ear with iodoform and boracic powder.

Behaviour of abscess cavity after evacuation.—When all sloughs and deleterious matter have been removed from the brain, the walls of the abscess cavity rapidly tend to come into apposition. The brain, which was previously compressed, bulges into and obliterates the space. This is especially so in acute abscess, the space being filled in a few hours. In very chronic abscess, on the other hand, the adhesion formed and the atrophy of brain tissue in its vicinity destroy or diminish its resiliency, and the cavity either remains as such for days or only very gradually becomes filled with expanded brain tissue.

Drainage of the abscess cavity.—After the evacuation of an acute abscess, drainage tubes are (for the reasons stated) of little value, provided the whole of the infective matter has been removed. If doubt exists on this point, a drainage tube is introduced. There can be no doubt that the pulsations of the brain cause, by striking against the tube, an irritation, which is increased if the tube be rigid. In chronic abscess the drainage tube may

be required. An absorbable decalcified chicken-bone drainage tube is introduced into the abscess cavity and stitched to the skin so as to retain it in position. Its inner margin is made to project just within the outer wall of the abscess cavity.

When the abscess cavity is still foul and pus formation is likely to proceed, an india-rubber, glass, or other non-absorbable drain is preferable, but it ought to be removed as soon as possible—twenty-four to forty-eight hours. In the event of an opening having been made between the tegmen antri or tympani and the abscess cavity, then the drainage is best conducted through it, and the opening through the squamous bone is allowed to heal.

Dressing the wound.—The exposed parts of the brain are well washed with an aseptic solution, dried, and dusted afresh with the iodoform and boracic powder. When the surgeon has succeeded in rendering the abscess cavity aseptic, the dura is lightly placed over the brain, but not stitched, leaving an exit for any discharge which may come to the surface of the brain, or for the drainage tube where such is used. When the cavity of the abscess still remains foul, the dura ought to be left reflected and iodoform gauze placed in contact with the brain, to secure quick absorption and to prevent the extravasation of infective matter in the membranes, which otherwise might occasion lepto-meningitis.

The osseous disc removed from the squamous may be re-implanted if there be a distinct probability of aseptic wound healing, and especially if the drainage has been carried through the tegmen. Otherwise the bone must be left out, as it undoubtedly would be shed in the presence of infective suppuration, and it might hinder the free escape of pus. Before the disc is re-implanted, the partial aperture made by the centre pin of the trephine is completed, and if the subject be over thirty years of age, or the bone be earthy, several small discs are removed from the larger one so as to leave a mere framework of bone. It is then introduced. The soft tissues are afterwards laid carefully over the bone and stitched.

Dressings.—When the dressings are to be placed in immediate contact with the brain, a thick layer of iodoform and boracic powder is dusted over it to prevent adhesion of the inner dressing; then some iodoform gauze and a pad of wood-wool with a slit in it, through which the ear is projected, the wool

lying close into the skull behind the ear. A second wood-wool pad is placed over the first, covering the ear, and then a layer of sublimated or other aseptic cotton wool is placed over the whole. These dressings are secured in their place by an aseptic gauze bandage.

Where the surgeon has succeeded in rendering the abscess cavity aseptic, the dressings are left for three weeks untouched, provided the temperature is normal and there is no stain through the dressings. At the expiry of that time they are removed, and the wound is either absolutely healed or there is a superficial granulating surface, according as the wound has been stitched or left open.

When the abscess cavity has been left infected, the dressings require to be changed daily and the cavity syringed out. In this case, boracic powder alone ought to be used instead of iodoform mixture, as the constant changing and renewing of the powder might induce the toxic effect of the iodoform. A great preventive against iodoform intoxication is the use of very large scales of iodoform instead of fine powder, as the latter is much more quickly absorbed.

Cerebellar abscess.—The skull over the mastoid is very thick, while over the inferior occipital fossa it is very thin.

Abscess of the cerebellum, arising from otitis media, is usually secondary to suppuration in the vicinity of the sigmoid sinus, and is often accompanied by thrombosis of that vessel. It seldom ensues from extension, by way of the internal auditory meatus, infective inflammation passing by this channel being much more frequently followed by lepto-meningitis. It is well to expose the sigmoid groove first, with the view of ascertaining the condition of the sinus. In such cases it is generally covered by granulation tissue, from which pus exudes. This ought to be removed along with the extradural pus. If there be an erosion in the dura at either side of the sigmoid, it would aid in indicating the locality of the fistula leading to the cerebellar abscess. Occasionally the pus from the cerebellum exudes by the inner side of the sinus, and it is possible, by extending the osseous incision inwards, to widen the aperture in the dura sufficiently to permit the free escape of discharge, and also to wash out the cavity. It is much better, however, to open the cerebellum from the outer and external aspect of the sinus, as the abscess can always be reached in this way, while the facilities for evacuation and manipulation are

greater, and the subsequent drainage much more under control. The manner of evacuation, washing, and drainage is similar in detail to that given under treatment of temporo-sphenoidal abscess.

In operating upon cerebellar abscess cases, it occasionally happens that respiratory difficulties are experienced. When this occurs the operation ought to be proceeded with rapidly. Altering the position of patient's head relatively to the trunk sometimes effects an improvement in the breathing. The anæsthesia ought to be as light as is consistent with quietness of patient. In two cases reported the respiration was suspended during the operation, and was maintained by artificial respiration in one for twenty-four hours, in the other for six, the heart meanwhile acting rhythmically. The operation was abandoned when the respiratory difficulties ensued. It would have been much better to have finished the operation while maintaining the respiration, as the evacuation of the large abscess might have saved the patient's life by relieving the pressure on the respiratory centre.

Abscess of the frontal lobes.—These abscesses may be opened either from the front of the brow or from the temporal regions, the choice being determined by the precise locality of the abscess. From the brow any abscess in the frontal may be reached, though those situated in the back part of the frontal would be nearer the surface of the temporal fossa. The skull in front of the brow is thicker than in the temporal fossa, but, in the operations through the latter, the temporal muscle has to be divided and separated, and its planes of fasciæ might be exposed to suppuration were they brought into contact with infective matter from the abscess. With proper precautions this ought to be avoided. The frontal sinus ought to be avoided in trephining from the brow, though in idiopathic frontal abscess the frontal sinus is frequently involved, and, if so, ought to be opened as a preliminary. Infection extending through the cribriform plate of the ethmoid may cause abscess near the mesial aspects of the frontal, and trephining through the frontal sinus and about the position of the middle line would suffice for the further search for pus. If the cribriform plate requires to be exposed, it is best done by removing a quarter-inch disc slightly above the glabella—remembering that the frontal lobes dip at this point to the level of the nasion.

Abscess in the base of third frontal convolution.—Abscess in the white matter near the base of the third frontal convolution may best be dealt with by approaching it through the temporal fossa. While digital pressure controls the temporal in front of the ear, an incision is made through the soft parts to the bone from a point three quarters of an inch behind the external angular process of the frontal, extending upwards and backwards for two and a half inches in an obliquely vertical direction, parallel with the fibres of the temporal muscle. The bone is then exposed by periosteal elevators, and the cut branches of the temporal artery are ligated. The centre pin of the trephine is placed on the bone at a point two inches behind the external angular process and an inch above its level. The pterion is a safe guide to the position, and obviates the necessity of measurements. The skull is thin at this place, and a few turns of the trephine are sometimes sufficient. The guard of the trephine ought to be placed about an eighth of an inch from its cutting edge. The middle meningeal artery has to be looked for. Occasionally it is situated in a canal in the bone, and will probably be cut while making this osseous incision. Its situation is near the pterion, usually grooving the anterior inferior angle of the parietal. By measurement it ought to be found from an inch and a quarter to an inch and a half behind the external angular process of the frontal, and about the same distance above the zygoma, so that operating as above, it must be exposed. When it is cut in the bone the osseous sulcus is stuffed with iodoform, and boracic powder pressed hard into the aperture by means of a fine probe. A small portion of fine catgut answers equally well. When it is exposed in the dura it ought to be ligated—a fine curved needle may be passed through the dura and a catgut ligature applied. Though this is the most direct route to the third frontal convolution, if it be deemed advisable to avoid touching the artery, a trephine disc may be removed half an inch in front or behind the pterion. The dura is then opened, and turned aside in the manner already described. The hollow needle, or sinus forceps, ought then to be inserted in a transverse direction into the white substance of the brain. The remainder of the proceeding for evacuation of the pus is similar to that described under temporo-sphenoidal abscess.

Opening of the frontal sinuses.—The frontal sinus is rudimentary in infants, small in children, increases in size about puberty, and

becomes fully developed at the age of twenty-five. The sinuses lie on either side of the middle line divided by a septum, which is frequently imperfect or perforated so that one sinus communicates with the other.

When both sinuses are involved, a vertical incision is made in the middle line, beginning at the root of the nose and extending upwards for an inch to an inch and a half. It penetrates all the tissues to the bone, and the periosteum is reflected to both sides by a periosteal elevator. A disc of bone from a quarter to half an inch in diameter is elevated from the middle line of the anterior wall so as to expose both sinuses. The osseous aperture may be extended to either side by means of the bur or by forceps. Access may be had by this incision to both sinuses. The infundibulum is made patent, the sinus washed, or if its mucous membrane has been destroyed and the cavity filled with granulation tissue, the latter requires removal. In the adult the frontal sinus is often prolonged outwards over the eyebrow, and as its floor is somewhat arched and irregular, it is difficult through the median incision to ensure the removal of deleterious products from its outer aspects. When an aperture exists in the upper eyelid, in connection with a fistula passing through the orbital roof into the frontal sinus, and when it is probable that the disease is confined to one sinus, the opening into it may with advantage be made to the outer side of the supra-orbital notch over the most prominent part of the sinus. A vertical incision here will be clear of the main trunk of the supra-orbital nerve, it will divide a few of the twigs of the frontal branch of the ophthalmic artery, which may be readily controlled by digital pressure against the sharp edge of the orbit, and any oozing from collateral vessels may be arrested by pressure on the temporal artery. At this point a quarter inch trephine is applied to the anterior wall of the sinus, and after the sinus has been exposed the probe ought to be introduced to ascertain the dimensions, form, and nature of the contents of the cavity. If its lining membrane has disappeared, and it is packed with granulation tissue, bathed in infective pus, the osseous opening is freely enlarged in an inward direction in line with the eyebrow, when the whole of the noxious contents can be removed, the cavity washed out, and the infundibulum rendered patent. Before closing the cavity, its posterior wall ought to be carefully inspected for erosions, and if any such be

found, they should be burred out and the dura mater examined. If extradural abscess be found, it should be dealt with as already described. The fistula, from the floor of the cavity to the orbital fossa and upper eyelid, is carefully cleansed by a sharp spoon, and a strip of gauze passed through the fistula. The frontal sinus is then stuffed with iodoform gauze, which is maintained until the cavity has become obliterated by a fresh connective tissue growth. The freer the osseous incision into the sinus, the quicker will the occlusion occur.

Treatment of the patient after evacuation of a cerebral abscess.—The patient is kept quiet, and his condition carefully observed by a nurse specially detailed and chosen for her superior intelligence and experience of such cases. Records of pulse, temperature, and respiration are continued after operation in the same way as before it.

After the tongue and mouth have been cleansed, milk diet or fluid nutriment is given in small quantities, at first peptonized, as the patient's digestion and assimilation are weak. The light fluid nutriment is continued for a fortnight or three weeks, and until the temperature is normal. A week after the operation, if the bowels have not been moved, a purgative, which will not cause vomiting, and which will give an easy motion without straining, is administered. The patient is kept in bed for from four to six weeks after the operation, however well he may be. In no case is he allowed to rise before the wound is quite healed—which is generally at the end of a fortnight or three weeks. If there be secondary abscess formation, there will be further delay in healing the wound, and also in permitting the patient to rise. It is better to exceed the period of quiet rest in bed than allow accidents to occur by permitting the patient to rise too soon.

The frequency of renewing the dressings depends on the case. Other things being equal, the more seldom the dressings are renewed the better for the patient, as when very frequent they are apt to disturb and fret him. If the abscess has been thoroughly evacuated, the wound rendered aseptic, and a decalcified drainage tube introduced, the dressings may be left for two or three weeks untouched, provided, there be no stain through them, the temperature, pulse, and respiration be normal, and the general condition of the patient be improving. Generally at the end of that period, on removing the dressing, the wound is found healed externally. When the abscess is imperfectly evacuated,

and the cerebral sloughs not removed, and when doubts are entertained regarding the asepticity of the wound, frequent dressings become imperative.

In *infective meningitis*, the sooner the source of the infection is removed, along with its path of communication with the meninges, the better. In some instances the disease may be so extensive that cure is impossible, but even in serious cases relief is often obtained by prompt operation. In several instances, where the symptoms were those of basal meningitis, and occasionally also in cases of cerebro-spinal meningitis, cure has followed eradication of the source of infection and removal of pus from the meninges. In middle ear disease, the antrum of the mastoid and its tegmen are removed when there are symptoms of infective meningitis of the middle fossa of the brain, and any purulent secretion existing between the skull and the dura or pia mater is washed out as previously described, and a similar operation is performed in the neighbourhood of the sigmoid sinus and the cerebellar fossa where the symptoms point to cerebro-spinal involvement.

In a case of cerebro-spinal meningitis, which has reached the suppurative stage, the prognosis is very hopeless, especially when the greater part of the cord is covered with pus. In such a case, after the cerebellar dura has been opened, pus oozes out for hours, a little at a time. Occasionally, however, the pus is inspissated and in the form of a layer retained by meshes of fibrin, which is very difficult to remove by irrigation. In cerebro-spinal meningitis, when the pus is still fluid, it has occurred to the author that single or multiple laminectomies, coupled with antiseptic irrigation from the cerebellar intradural space to the various openings in the spinal membranes, would be justifiable, and would give to an otherwise hopeless condition a prospect of relief, though such an operation has not yet been carried out. The irrigating antiseptic solution should be used warm.

CHAPTER VI.

RESULTS.

In the foregoing clinical record the cases which have been selected for publication are those which presented features of pathological or semiological interest, and they have for the most part been recorded *in extenso*, so as to form a contribution to the data from which conclusions may be ultimately formulated. This was felt to be especially necessary, as in consulting the scattered literature of the subject, the cases of intracranial pyogenic infective diseases recorded previously to the last few years were, for the most part, found to be fragmentary and defective.

Since 1886, however, there has been a change in this respect, and a series of cases of abscess of the brain which have been accurately diagnosed from the cerebral symptoms exhibited and carefully observed, have been reported by Dr. Gowers and Mr. Barker, Professor Greenfield and Mr. Caird, Professors Ferrier and Horsley, Professors Schede, Bergmann, and others; from the perusal of these much valuable information may be derived.

The cases reported in this volume are not arranged chronologically, but attention may be drawn to the fact, that the first cerebral abscess to be diagnosed accurately from the cerebral symptoms exhibited was that of a boy who had an abscess in the white matter in the vicinity of Broca's convolution (Case XXXIV.). That occurred in 1876, and though no operation was permitted during life, one was performed post mortem, when the abscess cavity was penetrated by the bistoury through a trephine opening. The second was in 1881 (Case XXX.). The abscess, diagnosed to be in the temporo-sphenoidal lobe, was operated on while the patient was *in extremis*. Though the patient died, this was the first case of cerebral abscess diagnosed

from localizing symptoms, and which was operated on during life. Five years afterwards Dr. Gowers and Mr. Barker had the honour of recording the first case successfully operated on, and others followed in quick succession.

The following is a résumé of the results obtained by the author, and they present the data upon which the foregoing observations have, for the most part, been founded. In the first group of cases presented, there has been great difficulty experienced in keeping in touch with the majority of the patients after their recovery from simple ablation of the mastoid. While many had the goodness to present themselves regularly as asked, others did so on perhaps one or two occasions and then ceased to attend. The statistics of the ultimate results, to this extent, have been impaired.

Fifty-four cases of ablation of the mastoid antrum and cells, with removal of infective material from the middle ear.—The author has in fifty-four cases of mastoid infective disease, extending from the middle ear and unaccompanied by intracranial lesions, ablated the antrum and mastoid cells, cleansed the middle ear, removing therefrom cholesteatomatous accumulations, masses of granulation tissue, and the ossicles when diseased. In none of these has the sigmoid sinus been opened purposely, nor has it in any case been wounded accidentally. The facial canal has generally been easily recognized and avoided—first, by its anatomical position relatively to the floor of the antral passage; and secondly, by its ivory-like casing forming a marked contrast with the cancellated tissue in its vicinity. But in one of these fifty-four operations, the facial was encroached on as it lay enclosed in a mass of granulation tissue. There had been very slight facial paralysis from the disease prior to the operation, which became marked after removal of the granulation tissue. The paralysis was greatly improved by the application of the continuous current and massage, commenced a few days after the operation and maintained for six weeks.

Though in the great majority of those radical mastoid ablations, the facial canal can easily be distinguished and avoided, yet when its relative position has been altered by pathological changes, and the nerve concealed thereby, the possibility of damaging it cannot be discounted.

In endeavouring to form an estimate of the value of this operation, data must be obtained on two points—first, its effect

on the cure of the otitis media; and second, its power to prevent infective invasion of the intracranial contents.

While judging of the effect upon the otitis media, the purulent discharge being taken as an index, consideration must be given to the extent of the disease and the particular direction of its invasion. It soon becomes manifest that these cases may be broadly divided into two categories, those in which the extension of the otitis media has been confined to the mastoid antrum and cells, and those in which it has also spread inwards and forwards into the petrous bone, the internal ear, and its recesses. The former are frequently purely pyogenic, while the latter are more often pyogenic inoculations upon an initial and old-standing tubercular base. Thirty-eight out of the fifty-four cases here referred to are comprised under the first category, and in them the disease was entirely eradicated by the operation, the otorrhœa was arrested, there being absolutely none when the patients were dismissed, and, as far as they could be kept under surveillance, they have remained in this state since. It is safe to assume that the majority of such cases were cured. On the other hand, when the disease involved the inner ear, and the petrosal recesses, difficult of access during operation, the discharge, though greatly lessened thereby, was in the majority of cases not absolutely cured. The otorrhœa often ceased for a time, but was prone to return on very slight provocation. No doubt in these the whole of the diseased tissue had not been eradicated, and from the insidiousness of the tubercular invasion, and the depth of its dissemination in many instances, it was beyond the limits of prudence to attempt its complete eradication. In such, a permanent opening through the mastoid affords not only free egress for discharge, but gives abundant facilities for maintaining the asepsis of the parts and for the removal of disintegrating material.

Regarding these cases from the second standpoint, the operation had enabled all of the fifty-four to have a mass of avascular connective tissue placed between the intracranial contents and the tympanum, forming an efficient barrier against pyogenic invasion of the middle and posterior fossæ of the skull, at their most vulnerable points. In this respect, therefore, the life of the patient was rendered more secure than before the operation against pyogenic invasion, even in those cases where the petrous bone still remained affected.

I. Statistical Table of Ablations of Mastoid Antrum and Cells for Pyogenic Otitis Media.

Disease.	No. Operated on.	No. Cured.	No. Relieved.
Purulent otitis media with extension limited to mastoid antrum and cells, - - -	38	38	
Purulent otitis media with extension, in addition to petrous bone and its recesses,[1]	16	5	11[1]
Total, - - -	54	43	11

Seventeen cases of infective pachy-meningitis externa, some with small extradural abscess, in which operations were performed. Seventeen recoveries.—Besides the five cases detailed in the clinical record under the heading of Extradural Abscess, there were seventeen in which, besides ablating the mastoid, the tegmen antri or tympani or the covering of the sigmoid sinus had to be removed, in consequence of pyogenic infection of the outer layer of the dura—pachy-meningitis externa. In these, the granulation tissue covering the dura and the infected pus, varying in quantity from a few drops to a couple of drachms, were removed. The extent of the tegmen or the sigmoid groove eradicated depended on the extent of the granulation tissue springing from the dura, sufficient bone being removed to expose, when possible, the limits of the pachy-meningitis. All these seventeen cases recovered.

Twelve cases of infective purulent lepto-meningitis, of which six were operated on and recovered—five of these were of the cerebral fossa and one of the cerebellar fossa.—Besides the cases which are given in detail in the clinical record, there were five which exhibited symptoms of purulent lepto-meningitis, at an early period, in which the tegmen was found eroded, the dura inflamed, softened and bathed in pus. After free incision of the inflamed membranes, purulent intradural exudation escaped, generally in drops, and continued to ooze for some time thereafter. In a sixth case, the dura on the external and posterior aspects of the sigmoid sinus was inflamed and of a yellow colour,

[1] In all these the discharge, though not actually stopped, is greatly reduced in quantity, and a connective tissue barrier has been formed between the diseased area and the more vulnerable intracranial regions.

from the layer of fibrinous pus in the inner membranes, which exuded slowly after incision. The symptoms and appearances in each of these six cases were indicative of lepto-meningitis, and in the last case the base of the cerebellar fossa was involved, the patient exhibiting signs of cerebro-spinal meningitis. These six cases recovered. Previous to these six cases there were six seen by the author, which were considered *at that time* as beyond hope. They were not operated on, and subsequently died.

Purulent lepto-meningitis is the most serious and rapidly fatal of the pyogenic infective brain diseases, and *à priori* was regarded by the author as almost hopeless—as it undoubtedly is hopeless when it becomes generalized. It was while of this opinion that the first of the above cases was operated on, and its recovery was regarded by him with surprise, succeeded by a doubt as to the accuracy of the diagnosis, notwithstanding the undoubted intra-membranous purulent exudation. The succeeding five cases were closely scrutinized before being operated on; their symptoms all pointed to lepto-meningitis of an infective kind, and the state of the membranes found on exposure confirmed the presence of purulent lepto-meningitis, at least localized. It is presumable that in those cases the membranes were in process of becoming generally infected, and the removal of the intra-membranous pathogenic focus arrested the further progress of the pial implication. These facts give encouragement for the future, as when purulent lepto-meningitis is recognized at its early stage and operated on without delay, recovery is probable. Even in cases where recovery is impossible, the operation is generally followed by a sense of relief experienced by the patient for a few days after the operation.

Six cases of cerebro-spinal lepto-meningitis, five of which were operated on with one recovery.—All of these cases are recorded in the foregoing chapters. There were six cases, five of which were operated on with one recovery. One of these which succumbed after operation was operated on in the author's absence. There was also a case showing symptoms of cerebro-spinal involvement, reported under the heading of abscess in the temporo-sphenoidal lobe, which recovered after operation, but this case is not included in the above five.

In view of the fact that one case of cerebro-spinal lepto-meningitis out of five recovered after operation, it is hoped that better results may yet be obtained, provided the cases are early

recognized and speedily operated on. In those that succumbed the post-mortem showed involvement of the whole cerebro-spinal system, in several instances pus being found spread over the base of the brain and the cord as far as the cauda equina. Recovery under such circumstances was not to be expected, and if from the clinical symptoms one could be certain that the purulent meningitis had involved the cord to such an extent, no operation limited to the cranium would be performed. But the symptoms of lepto-meningitis at an early period are so diffuse, and embrace such an extensive area, that *à priori* one would be apt to be deterred thereby from operating, though the purulent infection might yet be limited, and therefore amenable to operative treatment.

Twelve cases of recovery after operation for sigmoid sinus thrombosis.—Besides the cases reported in the chapter on Thrombosis—most of which were fatal—there were twelve cases of infective sigmoid sinus thrombosis which recovered after operation. In these the sigmoid sinus was freely exposed, the disintegrating contents removed, the sinus occluded, and the paths by which the infective matter travelled from the middle ear were eradicated. The tegmen tympani was always scrutinized in these cases for extensions of disease in that direction, and in several, multiple lesions were found requiring treatment. After these operations the recovery was as a rule rapid and simple.

To these may be added ten cases of infective sigmoid sinus thrombosis which are reported under various headings, of which seven recovered after operation, in which the sinus was ablated. Though these do not appear in the statistical table—in order to avoid double entries—yet, if they be added to the others, it gives twenty-eight cases with eight deaths.

Infective sinus thrombosis ranks, in order of fatality, next to suppurative lepto-meningitis. Whether a patient with infective sinus thrombosis will recover after operation depends on the stage reached by the infective invasion. If the operation be performed prior to general systemic involvement from the disintegrating thrombus recovery is probable. After the lungs have become the seat of infective infarction the hope of recovery from operation is distant, though in one case recovery succeeded an ablation of the sigmoid sinus when the patient had rusty-coloured expectoration and the lungs showed physical evidence of implication.

II. Statistical Table of Infective Intracranial Lesions.[6]

Disease.	No. of Cases.	No. Operated on.	No. Cured.	No. Died.
Pyogenic pachy-meningitis externa,	17	17	17	0
Extradural abscess,	5	5	5	0
Pyogenic cerebral lepto-meningitis,	12	6	6	6
Pyogenic cerebro-spinal lepto-meningitis,	6	5	1	5[1]
Superficial brain abscess and ulceration,	4	4	4[2]	0
Cerebral abscess—				
Temporo-sphenoidal,	10	9	8[3]	2
Frontal,	2	1	1	1
Parietal,	1	1	1	0
Cerebellar abscess,	8	4	4	4[4]
Meningo-encephalitis of frontal lobe,	1	1	0	1
Total necrosis of temporo-sphenoidal lobe,	1	1	0	1
Thrombosis of longitudinal sinus,	4	3	3	1
Thrombosis of cavernous sinus,	5	0	0	5
Thrombosis of sigmoid sinus,	18	17	13	5[5]
Total,	94	74	63	31

Twenty-five abscesses of the brain, of which nineteen were operated on and eighteen recovered.—With the exception of two cases of small abscess in the temporo-sphenoidal lobe which were operated on and which recovered, all the cases of cerebral abscess which have been directly under the author's care have been reported in detail in the chapter on Symptoms. There were ten cases of abscess in the temporo-sphenoidal lobe, of which nine were operated on and eight recovered. The one which died after operation was one of the earlier cases in which the whole

[1] One of the fatal cases was not under author's care.

[2] One case died seven weeks after operation from tubercular enteritis.

[3] One of these successful cases, though under author's care, was first operated on by Dr. Clark.

[4] Two of the fatal cases were not under author's care, and the other two were not operated on.

[5] In all the fatal cases lung infection and systemic toxæmia were present before operation. Sigmoid sinus thrombosis occurred as a complication in ten of the cases above tabulated under various other headings, and of these seven recovered after operation.

[6] Out of twenty-three cases of pyogenic intracranial extension from otitis media here recorded the lesions were on the left side in seventeen and on the right in sixteen.

temporo-sphenoidal lobe was destroyed and patient was operated on *in extremis*, the abscess having burst into the lateral ventricles. Besides these ten cases, there are four others recorded under the heading of Superficial Cerebral Abscess or Ulcerations of the Brain, which, had the pus from them been evacuated in the ordinary way, by trephining through the squamous portions of the temporal, would have undoubtedly been classed as ordinary cerebral abscess. Were these four added to the foregoing it would give thirteen abscesses in the temporo-sphenoidal lobe which have been operated on, with twelve recoveries.

Of the eight cerebellar abscesses recorded, two were not under the author's care, and other two were not operated on, leaving four that were operated on by the author, all of which recovered. One of these was a case of multiple abscess, one being in the cerebellum, while another was in the posterior portion of the temporo-sphenoidal lobe. This is, however, only once entered under the heading of Cerebellar Abscess. There are likewise two recurrent abscesses in the temporo-sphenoidal lobe each entered only once, though two abscesses were evacuated in each case, considerable intervals elapsing between the evacuation of each abscess.

III. Statistical Table of Intracranial Abscess.

Disease.	No. of Cases.	No. Operated on.	No. Cured.	No. Died.
Cerebral abscess—				
In temporo-sphenoidal lobe, -	10	9	8	2
In frontal lobe, - - -	2	1	1	1
In parietal lobe, - - -	1	1	1	0
Superficial (Ulceration of brain),	4	4	4	0
Cerebellar abscess, - - - -	8	4	4	4
Total, - - -	25	19	18	7
Extradural abscess, - - -	5	5	5	0
Total, - - -	30	24	23	7

Of the twenty-five cases of abscess of the brain mentioned in the table, there were nineteen operated on, with eighteen

recoveries. In these nineteen cases there were twenty-two abscesses evacuated, with twenty-one recoveries. One might almost conclude that in uncomplicated abscess of the brain, operated on at a fairly early period, recovery ought to be the rule.

Of extradural abscess (not including those recorded under Suppurative Pachy-meningitis) there were five, all of which recovered after operation. These five cases of extradural abscess, as well as the seventeen of purulent pachy-meningitis, may be regarded as the precursors of lepto-meningitis or cerebral abscess, and the arrest of the pathogenic processes by operation while yet extradural, prevented the graver intracranial mischief, which, in ordinary course, was sure to follow.

Under various headings, especially the record of Sigmoid Sinus Thrombosis, small abscesses are found which are not included in the above, as they were secondary and subordinate to the graver lesion, under which heading the case is placed. Many of these cases were very complicated, lepto-meningitis, abscess of the brain, as well as sigmoid sinus thrombosis and general toxæmia being present at the same time.

ADDENDUM.

SINCE this volume was in the press the following case of pyogenic infective lepto-meningitis with cerebellar abscess occurred, and as it pursued an unusual course, a *résumé* of it is here given as an addendum.

Résumé of a case of acute basal lepto-meningitis, with cerebellar abscess, œdema of the brain, and distension of the ventricles, arising from chronic otitis media purulenta. Operation. Recovery. Over two months subsequently rapid increase of the œdema of the brain, and sudden death.

J. B., aged 49 years, labourer, was admitted to the Western Infirmary, 21st February, 1893, in a comatose condition with marked respiratory difficulty.

History.—The items of history obtainable were meagre and imperfect. *Heredity.*—As far as can be ascertained the family history is good. *Personal.*—For forty years he has suffered from double otitis media purulenta, the result of measles at nine years of age. The amount of discharge has varied, but has always been greater on the left side. The amount from the right ear has been very slight during the last twelve months. Four weeks ago he had an attack of what his friends termed bronchitis, but in the light of what was found at the operation it might have been a pulmonary inflammation of an infective kind arising from the sigmoid sinus.

Present Illness.—Two weeks before admission he began to experience pain behind the left ear, extending to the frontal, and subsequently to the occipital regions. About this period he had three rigors. Six days before admission he was obliged to take to bed, and the following morning he vomited. He complained of pain and stiffness in his neck. Two days after this he fell into a lethargic condition, which state passed into one of unconsciousness, with incontinence of urine and marked respiratory difficulty.

Condition on Admission.—The patient lay in a semi-comatose condition, from which with great difficulty he could be roused to mutter a half articulate answer. His head was retracted on the neck. His pupils were equal, medium, and sluggish—almost fixed. There was an offensive odour from his breath, and his teeth were covered with sordes. He had marked periods of apnœa, varying from 20 to 75 seconds in duration, and brought to a close by a deep inspiration, the first of a series of four or five, after which a fresh pause occurred, which in turn was followed by a series of inspirations. This kind of respiration differed from the Cheyne-Stokes variety inasmuch as the inspirations were not graduated; they did not taper off at either end.

The lungs appeared normal, the heart and liver healthy.

The pulse varied much in frequency, being influenced by the breathing, but was quick, full, and easily compressed.

Temperature in the rectum, 100° F.

The left ear was filled with granulations and fœtid pus. The right was dry, though it contained old granulations. There was also pain and swelling in the course of the left internal jugular vein.

Diagnosis.—It was evident that the patient suffered from an interference with the respiratory centres in the medulla, most probably the result of lepto-meningitis of a purulent infective variety. As this resulted from pyogenic infection extending backwards, the sigmoid sinus was probably involved, and the cerebellum beyond was probably the seat of suppuration and ulceration. The brain was evidently œdematous from occlusion of basal veins by the meningitis, or from blocking of the fourth ventricle.

Operation (22nd February).—No anæsthetic was given as the œdema might have been thereby increased. The antrum of the left mastoid was opened and cleansed of its putrid contents. The sigmoid groove was exposed for fully a vertical inch, and about two drachms of pus were evacuated from the cerebellar fossa. The sinus was covered with pus and yellowish plastic exudation. Its external wall was softened, thick and partially disintegrated; its lumen was, however, still patent.

The opening over the cerebellar fossa was enlarged by the removal of two discs of bone from behind the sigmoid sinus. The cerebellar dura was covered with granulation tissue and pus. The inflamed and softened membranes were incised, and gave vent to a few drachms of pus, and pus continued to ooze slowly from the incision. The whole cavity was washed out and packed with iodoform gauze.

After the operation the patient's general condition greatly improved; he was restored to consciousness, and the respiratory difficulty was much lessened. It was observed that he could breathe normally when he lay on his right side (the side not operated on), whereas when he lay on his back or on his left side the characteristic breathing again showed itself. Ten days subsequently his respiration was so much improved that it was only noticed to be slightly affected during sleep. From this time forward he improved greatly. His intelligence returned, his appetite increased, and his wound underwent rapid cicatrization. About four weeks after

the operation he felt so well that he was clamorous to rise. He was allowed up in the sixth week, and was soon able to engage in various ward duties, and wished to be dismissed. He was, however, persuaded to remain under observation for two reasons—first, because he had still a sinus through the dura from which pus exuded, and second, because it was feared that the œdema of the brain and distension of the ventricles had not disappeared, though there was no physical evidence of their presence, and, if the latter of these two reasons were correct, he would be liable to sudden increase of the cerebral œdema.

On 28th April the patient, who had been as well as usual during the day, became sick and vomited during the night after he had gone to bed, and this was followed by some straining at stool. On walking back to bed he became giddy, and was shortly plunged into deep coma with marked respiratory difficulty, from which he died rather suddenly.

Post-Mortem Examination.—There was a portion of the dura mater extending backwards for a couple of inches from the sigmoid sinus which was black in colour, softened, and partly adherent in one place to a cicatrix of the soft structures outside of the skull which presented themselves through the trephine apertures. Attached to this blackened part of the dura mater there was a softened patch in the cerebellum communicating with a cavity in the cerebellar tissue containing inspissated pus. Unfortunately this escaped during removal and was not measured. There was evidence of an old-standing lepto-meningitis of the cerebellar fossa extending along the course of the pons. The layers of the inner membranes were adherent and injected. The basal veins and the venæ Galeni were surrounded by chronically inflamed membranes. The ventricles of the brain were greatly distended with serum, and the brain was markedly œdematous. The walls of the fourth ventricle were somewhat swollen and softened. It was clear that the death was due to a fresh accession of œdema.

INDEX.

ABERCROMBIE, case of abscess in medulla oblongata, 122.
case of lateral sinus thrombosis, 226.
Ablation of mastoid, results of, 327.
statistical table, 329.
Abscess of brain, 49.
absorption of contents, 109.
calcification of contents, 109.
character of pus in, 105.
character of surrounding brain tissue, 106.
course, 225.
diagnosis, 221.
differentiation from encephalitis, 221; from diffuse lepto-meningitis, 221; from infective sigmoid sinus thrombosis, 221; from tumour, 221.
encapsulation of, 107.
from contre coup, 58.
from sinus thrombosis, 236.
increase from within, 109.
influence on pulse and temperature by pressure, 114.
in white matter, 61, 72.
leakage or bursting of, 110.
may exert little pressure, 114.
mode of healing after evacuation, 123.
multiple generally pyæmic, 122.
parts most apt to be affected, 122.
pressure effects in relation to size, 112.
prognosis, with operation, 223; without operation, 224.
prognosis, danger of encysted abscess, 224.
remaining *in situ* while otitis media cured, 111.
results, 332.
size of, 113.
sloughs in, 115.
statistical table of results, 333.
symptoms, 130-150.
treatment after evacuation, 325.
Abscess, extradural, 49-57; subdural, 50.
Abscess in cerebellum, cases, 197-216.
operation for, 321.
results, 333.
symptoms, 194, 195.
Abscess in roof of fourth ventricle, case of, 216.
Abscess in middle cranial fossa, 80.
Abscess in occipital lobes, 194.
Abscess in parietal region, localizing symptoms, 193; case of, 193.
Abscess in temporo-sphenoidal lobe, cases of, 155-182.
localizing symptoms, 151.
operation on, 313, 314.
results, 332.
some pressure effects, 48.
Abscess of frontal lobe, case of, 183.
localizing symptoms, 183.
operation for, 321.
Abscess in vicinity of Broca's lobe, case of, 189.
operation for, 321.
Abscess of pons, symptoms, 195.
Abscess, subperiosteal mastoid, 64.
Abscess under deep cervical fascia, following sinus thrombosis, 265.
from suppuration in cerebellar fossa, 286.
from suppuration in mastoid cells, 287.
Abscesses of brain may empty themselves, 82, 110.
Absence of sensory paralysis in abscess of temporo-sphenoidal lobe, 152.
Acoustic apparatus, 2; nerve, 18.
Acute diseases of mouth, etc., in relation to sigmoid thrombosis, 261.
Acute lepto-meningitis, 84; from subdural abscess, 50.
Acute otitis media in relation to intracranial lesions, 55.
Acute periostitis of mastoid passing into abscess, rare, 64.
Acute processes, rare intracranial extension of, 62.
Acute purulent otitis media in relation to intracranial affections, 293.
Acute serous lepto-meningitis, 85.
appearances in, during operation, 85.
appearances in, post-mortem, 86.
localized, 85.
Acute squamo-mastoid periostitis, from cerebral abscess, 65.
Acute suppurative lepto-meningitis, consequence of bursting of abscess, 111.
Addenda to mastoid infective lesions, 84.
Admission of air into venous sinuses, 310.

Ætiology of cavernous sinus thrombosis, 245.
of infective thrombosis of sinuses, 231.
of intracranial pyogenic diseases, 52-63.
of longitudinal sinus thrombosis, 242.
of marasmic thrombosis of sinuses, 227.
of sigmoid sinus thrombosis, 260.
Albuminuria in sigmoid sinus thrombosis, 138.
Alternate hemiplegia in abscess of pons, 195.
Amyloid degeneration from chronic otitis media, 294.
"Anchoring" of brain, 123, 224.
Anæmic cerebral necrosis, arterial origin, 105.
Anæsthetics in inflammatory cerebral affections, 312.
Anorexia in abscess of brain, 138.
in infective thrombosis of sinuses, 238, typhoid type, 240.
Anterior condylar veins, 27.
Anterior temporal diploic vein, 30.
Antro-tympanal passage, 4, 10, 300.
Antrum mastoidium, 7.
Aperture between tympanum and glenoid fossa, 3.
Aperture into abscess cavity, 81.
Aphasia, in abscess of temporo-sphenoidal lobe, 153.
transient, in abscess of frontal lobe, 183.
Apyrexia in abscess of brain, 134.
Aqueductus Fallopii, more open in children, 84; pathogenic extension along, 74.
Arachnoid mater, 43; nerve supply of, 43.
Arachnoid villi, 45.
Arterial perivascular sheaths, infective extension by, 51.
Arterial supply of temporo-sphenoidal lobe, 18.
Arterial supply of tympanum, 5.
Arterial thrombosis and embolism, 51-72; in cortical arteries, 51; in relation to abscess in white matter of brain, 51.
Artery, ascending pharyngeal, meningeal branch, 33.
Artery, cerebral anterior, 18; cerebral middle, 18.
Artery, internal carotid, infective thrombosis of, 51, case of, 52; tympanic branches, 5.
Artery, internal maxillary, tympanic branch, 5.
Artery, middle meningeal, petrosal branch, 5.
Artery, occipital, posterior meningeal branch, 33.
Artery, posterior auricular, stylo-mastoid branch, 5.
Aspiration of sinuses, mechanism preventing, 33.
Attic of tympanum, 4.
Aural examination in abscess of brain, 142, 150.
Auricular branch of vagus (Arnold's nerve), 19.

Bacillus entericus, 54.
Bacillus meningitidis purulentæ (Neumann), 54.
Bacillus pyocyaneus (Gessard), 54.
Bacillus pyogenes fœtidus, 54, 55.
Bacillus tuberculosis, 54.
Barker, occurrence of optic neuritis in suppurative otitis media, 142; case of temporo-sphenoidal abscess, 326, 327.
Barr, case of thrombosis of internal carotid artery, 52; case of intracranial suppuration, pus seen with auriscope escaping through tegmen, 142.
Basilar meningitis from cavernous sinus thrombosis, 251.
Basilar purulent meningitis from infective sinus thrombosis, 236.
Bergmann, case of cerebral abscess, 326.
Bilateral paralysis, in abscess of pons, 195; in cerebellar abscess, 195.
Billroth, case of cavernous sinus thrombosis, 246.
Birmingham, measurements by, 9.
Bleeding from nasal septum, 47.
Blindness, in cerebellar abscess, 195; psychical, 155.
Blood in cerebellar fossa, effects of, 195.
Blunting of senses in abscess of brain, 132.
Blyth, hydraulic mechanism of jugular bulb, 38.
Böttcher, case of lung pigment in brain abscess, 56, 121.
Bouchut, on marasmic thrombosis of sinuses, 226; on significance of convulsions, 230.
Brachio-crural paralysis on same side as lesion in cerebellar abscess, 195.
Brachycephalic skull, 25.
Broca's lobe, abscess in vicinity of, 189.
Bur, rotatory, for opening mastoid, 296, 299.

Canal, carotid, 6.
facial, 14.
for tensor tympani, 5.
of Huguier, 6.
Canals, semicircular, *vide* Semicircular canals.
Capsule of abscess of brain, 107, 108; calcification of, 109.
Carbuncle of head, cause of cavernous sinus thrombosis, 246.
Carcinoma of middle ear, 128; cachexia in, 128; treatment of, 129; as cause of sigmoid sinus thrombosis, 261.
Caries, of mastoid cells, 67.
of petrous, as whole, 67.
of sphenoid, cause of cavernous sinus thrombosis, 246.
of temporal bone, most frequent situation of, 64.
Caries sicca, extensive in tympanum with little discharge, 68.
in relation to sigmoid thrombosis, 262.
tubercular, of base of skull, 67.
without perforation of membrana tympani, 294.
Cario-necrosis of skull, prevention of intracranial sequelæ, 292.
Carotid plexus of sympathetic nerves, 19.
Cavernous sinus, 29; anastomosis of, 29; in relation to internal carotid artery and to nerves, 33.

Cavernous sinus thrombosis, 245; cases of, 251; symptoms of, 247.
Cephalalgia in brain abscess, initiatory stage, 131.
in cavernous sinus thrombosis, 247.
in infective sinus thrombosis, 236, 240, 241.
in sigmoid thrombosis, 262.
Cerebellar abscess, operative treatment of, 320.
relation to sigmoid sinus, 74.
relative frequency of, 123.
results, 333.
size of, 113.
symptoms, 194.
Cerebellar arteries, emboli, 74; thrombosis of, 74.
Cerebellar fossa, abscess in discharging through posterior condylar foramen, 110; how attacked, 72.
Cerebellar veins, thrombosis of, 74.
Cerebellum, relations to posterior fossa, 18.
Cerebral abscess, *vide* Abscess of brain.
Cerebral abscess, true, formation of, 81.
Cerebral and cerebellar abscess in relation to infective sinus thrombosis, 236.
Cerebral cortex, involved in lepto-meningitis, 87.
Cerebral function, in infective sinus thrombosis, 236, 239.
Cerebral membranes, 40.
Cerebral otorrhœa, so-called, 110; case of true, 110.
Cerebral veins, 23.
Cerebration, slow, in brain abscess, 2nd stage, 133.
Cerebro-spinal fluid, 46.
Cervical abscess from venous thrombosis, 265.
Cervical lymphatic glands, 39.
adherent to jugular sheath, 39.
enlargement of, in tubercular otitis, 126.
enlargement of, in sigmoid thrombosis, 262, 265.
Channels of intracranial infection, 50.
Chemosis in cavernous sinus thrombosis, 247, 249.
Cheyne-Stokes respiration in brain abscess, 137; in cerebro-spinal lepto-meningitis, 196.
Chisel, separation of edges of fracture by, for removal of infective matter, 58.
Chiselling infected bone in head injuries, 292.
"Choked disc" in cavernous sinus thrombosis, 249.
Cholesteatomata, 55; micro-organisms in, 56; removal of, 327.
Chorda tympani nerve, 19; effects of stimulation and destruction of, 20; source of gustatory fibres of, 20.
Choroid veins, 23.
Chronicity of otorrhœa, no guarantee of safety, 55.
Chronic periostitis of mastoid, 65.
Circulation in brain abscess, initiatory stage, 132.
Clenching of teeth in cerebellar abscess, 194.
Clonic muscular contractions in infective thrombosis, meningeal type, 241.
Closure of gap in mastoid after operation, 302.
Clots in marasmic thrombosis, 229.
Cold, feelings of, in brain abscess, initiatory stage, 131.
Colour of pus in brain abscess, 105, 106.
Colour of face in brain abscess, 2nd stage, 139.
Coma, in brain abscess, 143; in infective sinus thrombosis, 240, 241.
Communications with tympanum, 5, 6, 7.
Condylar vein, anterior, 27; posterior, 26.
Congestion in marasmic thrombosis, 229.
Connective tissue barrier to pyogenic invasion after ablation of mastoid, 328.
Constipation in brain abscess, 2nd stage, 138.
in infective sinus thrombosis, 238.
Construction of intracranial sinuses in relation to thrombosis, 227.
Contracture, muscular, in marasmic thrombosis, 230.
Contre coup, brain injury by, 47; abscess of brain from, 58.
Convulsions in brain abscess, 139.
in children, 230.
in longitudinal sinus thrombosis, 243.
in marasmic thrombosis, 230; unilateral, 230.
prognostic value of, 230.
Cough in infective sinus thrombosis, 238.
COUPLAND, on cavernous sinus thrombosis, 226.
Course of cerebral abscess, 225.
Cranial percussion note, 146; "cracked pot," in extensive fractures, 149; value of, in cerebellar lesions, 196.
Cutis anserina in brain abscess, initiatory stage, 131.
Cure, apparent natural, of brain abscess, 144.

Danger of opening subarchnoid space at base of skull, 46.
Deep cervical glands, 39; affected from intracranial irritation, 39; cause "white swelling" of neck, 39.
Defect of ocular movements in cerebellar abscess, 195.
Deglutition in abscess of pons, 195.
Delirium in infective sinus thrombosis, 240, 241; in marasmic thrombosis, 230.
DEMONS, case of cavernous sinus thrombosis, 226.
Depression of fontanelles, 228, 231.
Desquamation of endothelium in infective sinus thrombosis, 232.
Destruction of chorda tympani during operation, 302.
Diagnosis, differential, between sigmoid thrombosis, brain abscess, and meningitis, 267.
of encephalitis, diffuse lepto-meningitis, general systemic infection, infective sigmoid sinus thrombosis, and cerebral tumour from abscess of brain, 221-223.
of infective sinus thrombosis, typhoid type from enteric fever, 240.
Diagnosis of marasmic thrombosis, 230.

Diarrhœa, exhausting, as cause of marasmic thrombosis, 228.
Diarrhœa, in infective sinus thrombosis, 238-240.
Differential cranial percussion note, 146.
in over-distended lateral ventricles, 148.
in rickety children, 147.
method of eliciting, 147.
Diminished sensibility, in abscess of pons, 195.
Diphtheria, extension to middle ear, causing sigmoid thrombosis, 56.
Diplococcus pneumoniæ (Frænkel) in subdural abscess, 54.
Diploic inflammation in relation to longitudinal sinus thrombosis, 242.
Diploic veins, 30.
Diplopia, cause of giddiness and headache, 21.
Discharge in tubercular otitis, 126.
Discoloration of sigmoid groove in infective sinus thrombosis, 234.
Disintegrating pneumonia, as cause of abscess of brain, 121.
Disintegrating thrombus in internal jugular and vena cava, 236.
Disintegration of lung in infective sinus thrombosis, 235.
Disintegration of thrombus in infective sinus thrombosis, 232; effects of, on bone and soft tissues, 234.
Distension of frontal and facial veins in cavernous thrombosis, 250.
Distension of superficial veins in longitudinal sinus thrombosis, 243,
Distension of ventricles in marasmic thrombosis, 229.
Drainage of brain abscess, 318.
Dressing antrum, etc., 306.
Dressing wound after opening brain abscess, 319.
Dual sinuses, liable to infective thrombosis, 231.
Dura mater, 40.
conical projection on, 72.
infective involvement of, 71.
nerve supply of, 48.
processes of, 42.
relation to skull, 41.
separation of, in injuries to skull, 57, 61.
Duration of cavernous sinus thrombosis, 251.
Duration of initiatory stage of brain abscess, 132.
Dyspnœa in infective sinus thrombosis, 238.

Eburnating sclerosis of temporal bone, 63.
Effusion into retro-ocular tissue in marasmic thrombosis, 229.
Elastic pad in jugular fossa, 35.
Emaciation in brain abscess, 2nd stage, 140.
Eminentia arcuata, 4.
Emissary veins, 25; measurements of orifices, 27, 28.
Emphysema from fracture through mastoid cells, 13.
Empyema of frontal sinuses, symptoms of, 151.
Encapsulated abscess of brain, case of, 108; increase of, 109.
Encapsulation of brain abscess, 107.
Encephalitis, differential diagnosis from brain abscess, 221.
Encephalitis from brain anchoring, 123.
from nitrate of silver injection into middle ear, 82.
zone of, round abscess, 10.
Encysted abscess of brain, 144, 224.
Enteritis, infective, specially in sigmoid thrombosis, 266.
Epilepsy, after traumatism with brain anchoring, 123; not after operation on abscess, 123.
Epistaxis in longitudinal sinus thrombosis, 243; in marasmic thrombosis, 230.
Equilibrium, disturbance of, in injury to semicircular canals, 21.
ERICHSEN, case of pus in cerebellar fossa discharging through mastoid foramen, 25.
Erosion and rupture of carotid artery from caries of petrous, 13.
Erosion, close to petro-squamosal suture, 72.
into carotid canal, 68.
into jugular fossa, 68.
into middle fossa, 67.
into sigmoid groove, 72.
of ossicles, 68.
of sigmoid groove, from infective sinus thrombosis, 234.
ERSKINE, case of extensive disease of tympanum without perforation of membrane, 63.
Erysipelas, cause of infective sinus thrombosis, 231.
of face and nose, cause of cavernous sinus thrombosis, 246.
of scalp, cause of infective longitudinal sinus thrombosis, 242.
Eustachian tube, 4, 5, 11, 12.
abscess in wall of, 84.
escape by, of discharge in tubercular otitis, 126.
escape of pus from pharyngeal orifice, 53.
swallowing discharge from, 266.
Evacuation of brain abscess, effect on pulse rate, 136; natural, 224.
Evidence of intracranial abscess, by auriscopic examination, 150; from quantity of aural discharge, 150.
Excitement in infective sinus thrombosis, 241.
Exhaustion, death by, in infective sinus thrombosis, 240.
Exophthalmos in cavernous thrombosis, 247; in marasmic thrombosis, 229.
Expansion of brain after abscess evacuation, 123.
Expectoration, infective, swallowing, 267.
Expectoration in infective sinus thrombosis, 239.
Exploratory cannula for brain abscess, 316.
Exposure of mastoid cells by operation, 301.
Exposure of sigmoid sinus by operation, 307.
Extension of thrombus to veins in infective sinus thrombosis, 233.

External auditory osseous meatus, 2.
relation to carotid canal, 3.
relation to mastoid antrum, 7, 9.
relation to floor of middle fossa, 10.
External hydrocephalus, 47, 85.
External pachy-meningitis, 49.
Extracranial abscess from infective sinus thrombosis, 234.
Extracranial venous obstruction and distension in marasmic thrombosis, 230.
Extradural abscess, 49, 57, 58, 60, 61, 69.
from frontal sinus suppuration, 324.
from infective sinus thrombosis, 233.
in cerebellar fossa, 73.
over tegmen, treatment of, 312.
Extradural escape of pus from subdural abscess, 75.
Extradural extravasation of blood, 57.

Facial, or Fallopian, canal, 4, 6, 11.
avoidance during operation, 300.
compact bone surrounding, 16.
course of, 14.
depth from surface of mastoid, 15.
geniculate ganglion in, 14.
in infant, 15.
relation to antro-tympanal passage, 15.
relation to fenestra ovalis, etc., 15.
relation to fenestra rotunda, 16.
relation to tympanum, 14.
risk in removing granulations from, 300.
Facial hemiatrophy from paralysis of facial nerve, 20.
Facial nerve, 18; paralysis of, 19.
Facial paralysis, differentiation between central and peripheral, 20, 153.
in carcinoma of middle ear, 128.
in children, 15.
in tubercle of middle ear, 124.
of central origin in temporo-sphenoidal abscess, 153.
Facial twitchings during operation, 300.
Facial veins and pterygoid plexus, 33.
FENMAN, calcification of brain abscess contents, 109.
Fenestra rotunda, 4.
Fenestra ovalis, 3.
FERRIER, case of cerebral abscess, 326.
Fibrous obliteration of sinus in infective sinus thrombosis, 237.
Floor of middle fossa, level of, 9, 18; peculiar relation to antrum, 18.
Floor of tympanum, 4.
Fluctuation in brain abscess, 315.
FLÜGGE, on cocci in jugular vein from diphtheria of tonsil, 56.
Fœtal distribution of intracranial sinuses in relation to aspiration, 38.
Fœtid bronchitis as cause of abscess of brain, 121.
Fœtid pericarditis as cause of abscess of brain, 121.
Fœtor, of cerebral abscess, etc., 106.
of expectoration in infective sinus thrombosis, 239.
of stools in infective sinus thrombosis, 239.
many non-pathogenic organisms produce, 55.
Fœtus, tympanic mucosa in, 11.

Foramen, anterior condylar, 27.
mastoid, 26.
posterior condylar, 26.
Foramen of Majendie, 45; effects of closure of, 46.
Foramen spinosum, 18.
Foramina of Key and Retzius, 45.
Formative process in sinus wall in infective sinus thrombosis, 235, 268.
Fossa subarcuata, 6, 11, 14.
Fracture of base of skull causing cavernous sinus thrombosis, 246.
Fracture of frontal sinus, causing extradural abscess, 60.
Fractures of skull, case of lepto-meningitis from, 57; compound, 57; punctured, dangers of, 57.
Frontal diploic vein, 30.
Frontal lobes, abscess in, 183.
cases of, 183.
operative treatment of, 321.
orbital surface liable to injury, 44, 47.
Frontal sinuses, exposure of, for suppuration within, 292.
suppuration, operative treatment, 322.
surgical anatomy of, 22.
symptoms of empyema of, 151.

GAIRDNER, case of thrombosis of internal carotid artery, 52.
Gangrene of lung in sinus thrombosis, 235, 239.
Geniculate ganglion, 14.
Giddiness from diplopia, 21.
Giddiness from injury to semicircular canals, 21.
"Girdle" pains in spinal lepto-meningitis, 241.
Glandular enlargement in carcinoma of middle ear, 128.
in sigmoid thrombosis, 262.
in tubercle of middle ear, 124, 126.
Glenoid fossa, guide to level of base of brain, 9.
Glosso-pharyngeal nerve, tympanic branch, 19.
GOWERS, relative frequency of abscess in different parts of brain, 123.
case of abscess, with latent period of 20 years, 144.
case of temporo-sphenoidal abscess, 326, 327.
Gouge for mastoid operations, 296, 303.
Granulations from dura, protective, danger in abrupting, 68, 75, 235, 301.
Granulations, removal from sigmoid sinus, 320.
Great petrosal nerve, 19.
Green discoloration of bone and soft tissues, 68.
Greenish colour of brain abscess pus, 106.
GREENFIELD AND CAIRD, case of temporo-sphenoidal abscess, 153.
GRUBER, on intracranial extension of inflammation along fossa subarcuata, 14.
on case of true otorrhœa cerebralis, 110.
on case in which brain abscess remained *in situ* while otitis media became cured, 111.

GUTHRIE, case of rupture of carotid artery from fracture of petrous, 13.

HABERMANN, case of tuberculosis of tympanum and mastoid cells, without perforation of membrane, 63.
Hæmorrhage, absent in disintegrating sinus thrombosis, 73.
cerebral, in cavernous sinus thrombosis, 251.
into abscess cavity, 114.
into subdural space, prevention of, 42.
Hæmorrhagic extravasations into brain substance, 51, 81.
Hæmorrhagic necrosis of brain—venous origin, 105, 114.
Headache from diplopia, 21.
in brain abscess, 131, 132.
in marasmic thrombosis, 230.
in sigmoid thrombosis, 262.
Headache persistent, and vomiting, significance of, 138.
Hearing, acuteness lessened in tubercular otitis, 126.
Hemiatrophy of face in facial paralysis, 20.
Hemiplegia, alternate in abscess of pons, 195.
in cavernous sinus thrombosis, 251.
in large temporo-sphenoidal abscess, 152.
Hepatic abscess in infective sinus thrombosis, 239.
Hepatic pain in infective sinus thrombosis, 239.
HEWETT, case of infective thrombosis with lung involvement recovery, 268.
HORSLEY, case of cerebral abscess, 326.
HUGHLINGS JACKSON on "dream state," 154.
HULKE, case of cavernous sinus thrombosis, 246.
Hydrocephalus, external, 47, 85; internal, 85.
Hyperacuteness of senses in infective sinus thrombosis, 241.
Hyperakusis in facial paralysis, 20.
Hypertrophic sclerosis of antrum, 299.

Importance of convulsions in cerebral localization, 139.
Incision for opening antrum, 297.
Incoordination in cerebellar affections, 195.
Increase of encapsulated abscess, 109.
Incubator, inflamed tympanum, a model, 52.
Indentation of brain by conical thickening of membranes, 80.
Indications for opening mastoid antrum, 294.
Infancy, marasmic thrombosis in, 227; sigmoid thrombosis seldom in, 260; temporal bone in, 11.
Infarctions, pulmonary, in sinus thrombosis, 235, 239.
in sigmoid thrombosis, 266.
symptoms of, 239, 266.
Infective cerebro-spinal lepto-meningitis, results of operations, 330; amenable to operative treatment, 331.
Infective embolism, cause of cerebral necrosis, 121; cause of pyæmic abscess, 121.
Infective enteritis in sigmoid thrombosis, 266.
Infective extension from tympanum, 67.
Infective fractures of skull, 57.
Infective inflammation on outside of skull and its intracranial effects, 49.
Infective injury of skull without fracture, 61.
Infective intracranial extension, through sutures, 72; without visible apertures, 72.
Infective intracranial invasion, from frontal sinuses, case of, 288; without visible wound, case of, 289.
Infective intracranial lesions, statistical table of, 332.
Infective involvement of membranes and brain, 71.
Infective involvement of middle fossa, 71.
Infective lepto-meningitis, treatment, 313, 325.
Infective lesions, causing thrombosis of cavernous sinus, 246, 247.
causing thrombosis of longitudinal sinus, 242.
causing thrombosis of sigmoid sinus, 261.
Infective necrosis of skull, 61.
Infective or inflammatory sinus thrombosis, 231.
ætiology of, 231.
pathology of, 232.
symptoms of, 236.
Infective otitis media, 62.
Infective pachy-meningitis externa, results of operations, 329.
Infective pneumonia in infective sinus thrombosis, 239-267.
Infective punctured wounds of bone, effects of, 56, 57.
Infective purulent lepto-meningitis, results of operations, 329; probable recovery if early operation, 330.
Infective pustules in nose, treatment of, 293.
Infective sigmoid sinus thrombosis, 261; fatality of, 331.
results of operations, 331.
symptoms, 262.
treatment of, 307.
with multiple additional lesions, 331.
Infective wounds of orbit, treatment of, 293.
Inferior petrosal sinus, 30; in jugular foramen, 33; in relation to jugular bulb, 37.
Inflammatory necrosis of brain, 114.
Inflammatory zone round brain abscess, 106.
Infra-orbital neuralgia in thrombosis of cavernous sinus, 247.
Initiatory stage of abscess of brain, 130.
Injecting fluid from antrum, caution in, 305.
Injecting fluid through apertures in tegmen, caution in, 313.
Injection of fluid into middle ear, effects of, 21.
Injections into subdural space, caution with regard to, 42; effects of, 43.
Injuries leading to intracranial disease, 56.

Instruments for opening antrum, 296.
Intelligence in infective sinus thrombosis, 240.
Internal antiseptics, administration of, 312.
Internal carotid artery, thrombosis of, from otitis media, 52.
Internal hydrocephalus, 85.
Internal jugular vein, 32; extension of thrombosis to, 233, 265; ligation of, 310; pain on pressure over, 265.
Internal maxillary veins, 33.
Intracranial abscess, statistical table, 333.
Intracranial effects of sinus thrombosis, 233.
Intra-jugular process, 33.
Iodoformization of wound prior to evacuating pus, 315.
Irascibility in infective sinus thrombosis, 211.

Jacobson's nerve, 19.
JORDAN-LLOYD, case of cavernous sinus thrombosis, 246.
Jugular bulb, in relation to inferior petrosal sinus, 37; in relation to sigmoid sinus, 36.
Jugular foramen, compartments and contents of, 32.
Jugular fossa, perforations of, 7.
Jugular sheath, relation to enlarged glands, 39.
Jugular veins, effect of respiration on, 34.

KATZ, on diphtheria extending to middle ear, 62, 261.
Knee of sigmoid groove, 16.
KÖRNER on relation of floor of middle fossa to external auditory meatus, 10; on thickness of osseous external wall of sigmoid groove, 17.
KNAPP, on cavernous sinus thrombosis, 226.

Labyrinth in caries of petrous, 67.
LANCIAL, on sinus thrombosis, 227.
Languor in infective thrombosis,
LAPERSONNE, on cavernous sinus thrombosis, 226.
"Latent period" in brain abscess formation, 149.
Lateral sinus appearance in section, 17.
Lateral sinus groove, 17; surface guide, 17.
Lateral sinus in relation to cerebellum, 18.
obstruction of, 32.
Leakage of brain abscess, 111; symptoms of, 143.
LEBERT, on convulsions in cerebral abscess, 138.
LEIDY, on petrous bone, 13.
Lepto-meningitis, acute, 50.
constipation in, 138.
pathology of, 84.
pulse quick small in, 135.
respiration quick in, 137.
vomiting in, 138.
Lepto-meningitis, acute purulent, from rupture of brain abscess, 111.
Lepto-meningitis, acute serous, case of, with external hydrocephalus, 86.
pathology of, 85.
Lepto-meningitis, cerebro-spinal, *vide* Infective cerebro-spinal lepto-meningitis.
Lepto-meningitis by infective extension along nerve sheaths, 52, 67, 74.
Lepto-meningitis, diffuse, diagnosis of, from brain abscess, 221.
Lepto-meningitis, from abruption of granulations, 235.
from erysipelas in old people, 62.
from infective extension by aqueduct of Fallopius, 74.
from infective thrombosis, 234.
from leakage of brain abscess, 111; symptoms, 143.
from otitis in young children, 84.
from tubercular caries, 67.
in meningeal type of infective thrombosis, 241.
of cerebellar fossa, localizing symptoms, 194.
purulent cerebro-spinal, cases of, 88, 102.
purulent, cortex involved in, 87; often encephalitis and abscess in, 87; pathology of, 87.
Lepto-meningitis, purulent, *vide* Infective purulent lepto-meningitis.
Lepto-meningitis, resemblance of, to meningeal type of infective thrombosis, 242.
spinal, symptoms of, 241.
suppurative from frontal sinus suppuration, case of, 292.
suppurative, of cerebellar fossa, respiration in, 137; treatment of, 313.
tubercular, 125.
Leptothrix buccalis in pus from mastoid antrum, 54.
Leucocytal exudation in brain abscess, 81.
Life, increased security of, from ablation of mastoid, 328.
Ligation of internal jugular for infective sigmoid thrombosis, 310.
Liver, infarctions of, in infective thrombosis, 236.
Local lesions and inflammatory thrombosis, 231.
Localities of venous thrombosis, 227.
Localizing symptoms in brain abscess, 145.
of cerebellar abscess, 194.
of frontal abscess, 183.
of parietal abscess, 193.
of temporo-sphenoidal abscess, 151.
Location of marasmic thrombosis, 227.
Longitudinal sinus, frequency of marasmic thrombosis of, 228.
superior, 24.
Longitudinal sinus thrombosis, 242.
symptoms of, 243.
infective, cases of, 244.
marasmic, case of, 243.
Lung, gangrene and abscess of, 239; infarctions of, in infective thrombosis, 235.
Lung pigment in brain abscess, 56, 121.
Lymphatics, cephalic, 38.
Lymphatic vessels as channels of infection, 52.
Lymphatic vessel with veins of Galen, 39.

Mallet and chisel, objections to use of, in exposing mastoid antrum, 303.
Malleus, 5.
M'Conville, case of cerebellar and temporo-sphenoidal abscess, 122.
Marasmic cavernous thrombosis never primary, 245.
Marasmic sigmoid thrombosis, by extension, 261 ; prognosis in, 268.
Marasmic thrombosis, 227.
ætiology of, 227.
affects azygos sinuses, 227.
location of, 227.
pathology of, 228.
points of difference from infective thrombosis, 236.
symptoms of, 229.
Mastication in facial paralysis, 20.
Mastoid, ablation of, *vide* Ablation of mastoid.
Mastoid abscess, subperiosteal, 64; cases of, 66.
Mastoid antrum, 7.
closure of gap after ablation of, 302.
depth from surface, 7, 299.
guide to position of, 9, 297.
operation to expose, 297.
relation to floor of middle fossa, 9, 300.
relation to orifice of external auditory osseous meatus, 9.
relation to posterior wall external auditory osseous meatus, 297.
Mastoid cells, 6.
development of, 12.
escape of pus from, into digastric fossa, 287.
exposure of, 301.
relation of, to sigmoid sinus, 13, 72.
Mastoid foramen, pus from, 26.
Mastoid inflammation in sigmoid thrombosis, 262.
Mastoid, necrosis of, 84.
Mastoid process, 12; double, 13; separate, 13.
Mastoid redness and swelling in brain abscess, 143.
Masto-squamosal suture, 5, 7, 8.
at birth, 11.
escape of pus by, 65.
persistence of, 8.
surgical importance of, 9.
Mastoid vein, 25 ; thrombosis in, 25 ; overdistension of, 263.
Maxillary inflammation as cause of cavernous thrombosis, 246.
Measly rash in infective thrombosis, 240.
Measurements of condylar and mastoid foramina, 27, 28.
Meatus, external auditory osseous, 2, 3, 5, 10, 16.
Mechanism preventing aspiration of sinuses, 33 ; unaffected by change of position of head, 38.
Medulla oblongata, abscess in, 122 ; involved in meningitis, 267.
Membrana tympani, caries sicca without perforation of, 294.
extensive otitis without perforation of, 63.
obliquity of, in infancy, 3.
Membrana tympani, perforation of, and access of micro-organisms, 52.
Membranes, cerebral, 40 ; translucent, in child, 42.
Meningeal hæmorrhage from marasmic thrombosis, 229.
Meningeal type of infective sinus thrombosis, 241 ; symptoms of, 241.
Meningitis and brain abscess, 49.
Meningitis, association with infective thrombosis, 231, 234.
basilar, with cavernous thrombosis, 245, 251.
cerebro-spinal, from rupture of cerebellar abscess, 111.
cerebro-spinal, purulent in sigmoid thrombosis, 267.
in sigmoid thrombosis, 266.
tubercular, 67, and otitis, in infants, 84.
Meningo-encephalitis, case of, 102.
Mental obscuration in brain abscess, 134.
Meteorism in infective thrombosis, 240.
Method of incising dura mater, 315.
Method of rendering scalp wounds, etc., aseptic, 291.
Micrococci in cerebral abscess, 106.
Micro-organisms in cholesteatomata, 55.
Micro-organisms, infection with, through perforated membrana, 52.
Micro-organisms in intracranial pyogenic disease, 52.
in mastoid, 53.
in neglected otorrhœa, 53.
of naso-pharynx, access of, to tympanum, 52.
spread from tympanum to naso-pharynx, 53.
Middle cerebral vein, 23, 29.
Middle compartment of jugular foramen, 33.
Middle cranial fossa, abscess in, 80.
level of floor of, 9, 18.
relation of, to external auditory meatus, 10.
relation of, to mastoid antrum, 9, 18.
Middle ear, inflammation of, *vide* Otitis media.
tubercle of, 124.
carcinoma of, 128.
Middle fossa, abscess in, 80.
Middle meningeal veins, 23, 33.
Miller, case of empyema of frontal sinuses, 288.
Mind, in meningeal type of infective thrombosis, 241.
in sigmoid thrombosis, 266.
Minute emboli in white matter, 72.
Minute sloughs over abscess wall, 106.
Mode of answering in meningeal type of infective thrombosis, 241.
Mode of infective extension from tympanum, 67.
Monoplegia in cavernous thrombosis, 251.
Motor effects of facial paralysis, 20.
Mucous membrane of tympanum, 4; extension of disease from naso-pharynx to, 62.
Multiple abscess of brain, 114, 122.
Multiple trephining in extradural abscess, 61.

Muscle, stapedius, 6.
Muscular fibrillæ in facial paralysis, 20; in brain abscess, 141.
Muscular jerkings in sigmoid thrombosis, 267.
Mydriasis in brain abscess, 145; in cavernous thrombosis, 250; in frontal lobe abscess, 183.
Myoidema in brain abscess, 141.
Myosis in brain abscess, 145.
in cavernous thrombosis, 250.
in frontal lobe abscess, 183.
from lesion of pons, 195.

Nasal septum, bleeding from, 47.
Nasal veins, in relation to cavernous sinus, 29.
Nasal ulceration cause of infective thrombosis, 231, 246.
Nausea, vomiting apart from, 138.
Necrosis of brain—anæmic, hæmorrhagic, ætiology of, 105, 114.
Necrosis of brain from occlusion of large arterial trunk, 52, 105.
Necrosis of mastoid in subperiostal abscess, 64.
Necrosis of mastoid, etc., in children, 84.
of skull, in relation to intracranial disease, 61.
of tegmen, sigmoid groove, etc., 69.
of temporal bone, most frequent situation of, 64.
total, of temporo-sphenoidal lobe, case of, 115.
Needle, hollow, in exploring for pus in brain, 316.
Nerve, Arnold's, 7; Jacobson's, 6.
Nerves passing through temporal bone, 18.
acoustic, 18.
Arnold's, 19.
carotid, sympathetic plexus, 19.
chorda tympani, 19.
facial, 18.
Jacobson's, 19.
large superficial petrosal, 19.
small superficial petrosal, 19.
to stapedius, 19.
tympanic plexus, 19.
Nerve sheath and subarachnoid space, 45.
Nerve symptoms in cavernous thrombosis, 250.
Nerve, 3rd, in cavernous thrombosis, 250; in temporo-sphenoidal abscess, 154.
Neuralgic pain in cavernous thrombosis, 247.
Nystagmus, from injury to semicircular canals, 21.

Obliquity of posterior wall of external meatus, 297.
Obliteration, of abscess cavity after evacuation, 123.
pathological, of mastoid antrum, 299.
of sigmoid sinus by operation, 309.
Obstruction, thrombic, of jugular bulb, 32.
of knee of sigmoid sinus, 32.
of lateral sinus, 32.
of lower part of sigmoid, 32.
Occipital diploic vein, 3.
Occipital lobes, abscess of, 194.
frequent seat of pyæmic abscess, 123.
Occipital lymphatic glands, 38.
Occipital sinus, 30,
Occiput, thinness of, 320.
Occlusion of abscess cavity after operation, 318.
Ocular paresis in abscess of pons, 195.
Ocular pressure symptoms from intramembranous injection of fluid, 43.
Odour from breath in brain abscess, 140.
Odour from otorrhœa, etc., in sigmoid thrombosis, 266.
Odour in relation to virulence of otorrhœa, 55.
Œdema, extracranial, in infective sinus thrombosis, 236.
Œdema, in brain abscess, 81.
in red softening, 102.
of anterior fontanelle in longitudinal sinus thrombosis, 243.
of brain in marasmic thrombosis, 229.
of eyelids in cavernous thrombosis, 247-249.
of mastoid region in sigmoid thrombosis, 263.
of pharynx and tonsil in cavernous thrombosis, 249.
of scalp in longitudinal sinus thrombosis, 243.
of side of face in cavernous thrombosis, 249.
of tissue round abscess, 106.
Oidium albicans in brain abscess, 56, 122.
Opening, double, into abscess cavity, 318.
Opening tympanic attic, 301.
Operation for abscess near Broca's lobe, 322.
for cerebellar abscess, 320.
for exposing frontal sinuses, 322.
for frontal abscess, 320.
for opening mastoid antrum, 296; points in conformation of bone, 297.
for temporo-sphenoidal abscess, 313, 314.
to expose sigmoid sinus, 307.
Ophthalmic veins, anastomosis of, 29.
Ophthalmoplegia in cavernous thrombosis, 250.
Opium poisoning, resemblance of second stage of brain abscess to, 134.
Optic neuritis, disappearance after operation, 224.
in brain abscess, 141.
in cerebellar abscess, 195.
in tympanic suppuration, 142.
Optic thalamus, abscess in, 122.
Orbital cellulitis cause of infective sinus thrombosis, 231.
Orbital cellulitis in longitudinal sinus thrombosis, 243.
Orbital fistula, 324.
Orbital inflammation cause of cavernous thrombosis, 246; diagnosis from cavernous thrombosis, 247.
Orbital pulsation in cavernous thrombosis, 251.
Orbital purulent collections result of cavernous sinus thrombosis, 234.
Osseous erosion in sigmoid thrombosis, 261.
Ossicles, tympanic, 5; removal of, 302.
Ossification of temporal bone, two forms, 2.

Otalgia in brain abscess, 131 ; in sigmoid thrombosis, 262.
Otitis media, acute, prevention of intracranial infection from, 293.
Otitis media, cases of, two categories from operative standpoint, 328.
cause of subperiosteal mastoid abscess, 65.
cause of infective sinus thrombosis, 231.
cause of sigmoid thrombosis, 260, 261.
chronic, treatment of, 293.
escape of pus from Eustachian tube, 295.
primary acute, rarely intracranial sequelæ from, 55.
rarely involves semicircular canals, 21.
relative to intracranial inflammation, 62.
results of operations, 327.
statistical table of ablations of mastoid for, 329.
Otitis media purulenta, cause of cerebellar and temporo-sphenoidal abscess, 122.
existence of, without perforation of membrane, 62.
in children, as cause of lepto-meningitis, 84.
with initiatory symptoms of brain abscess, 132.
Otorrhœa cerebralis, 110.
Otorrhœa, chronic, 294.
chronicity of, no guarantee of safety, 55.
neglected, micro-organisms in, 53; odour no indication of virulence, 55.
Over-distension of emissary veins in sigmoid thrombosis, 263.
"Overflow incontinence" in brain abscess, 138.

Pacchionian bodies, inflammation of, 242.
Pachy-meningitis externa, 49, 301.
results of operations for, 329.
Pachy-meningitis hæmorrhagica, 42.
Pain, abdominal, in infective sinus thrombosis, 240.
cervical, in infective sinus thrombosis, 240 ; in sigmoid thrombosis, 264.
in brain abscess, 2nd stage, 132.
in brain abscess occasionally absent, 132.
in carcinoma of middle ear, 128.
in cavernous thrombosis, 247.
in cerebellar abscess, 195.
in red softening, 102.
on percussion over mastoid in sigmoid thrombosis, 264.
Pains in limbs in brain abscess, 132.
PANAS, case of sinus thrombosis from gangrene of tonsils, 227.
"Papering" mastoid wound, 306.
Papulated eminence of membranes, 80.
Paralysis, facial, of central origin, 153.
Paralysis in brain abscess, 139.
in cerebellar abscess, 195.
in temporo-sphenoidal abscess, sequence of, 152.
Paralysis of facial nerve, in children, 15, 84.
in carcinoma of middle ear, 128.
in tubercular otitis, 124.
results of, 19.
Paralysis of nerves in cavernous thrombosis, 247.
Paralysis of third nerve in temporo-sphenoidal abscess, 154.
Paresis in infective sinus thrombosis, 241.
Parietal region, abscess in, case of, 193 ; localizing symptoms, 193.
Parieto-squamo-mastoid junction, 13.
Parotid lymphatic glands, 38.
Partial thrombosis of sigmoid sinus, 267.
PASSET on bacillus pyogenes fœtidus, 55.
Pathogenic affections of scalp and face in relation to intracranial disease, 61.
Pathogenic extension, along vessels, 72.
by aqueduct of Fallopius, 74.
to subdural space, 74.
Pathological conditions leading to intracranial disease, 56.
Pathological evacuation of brain abscess, 144.
Pathology of brain abscess, 49.
of infective sinus thrombosis, 232.
Patient, preparation of, for operation on mastoid, 295.
Patients in aural hospitals, age of, 294.
"Peasoup" stools, in infective sinus thrombosis, 240.
Pendulum movements, in injury to semicircular canals, 21.
Percussion note, cranial, 146, 147.
Perforation, osseous, frequent seats of, 68; of sigmoid sinus, 73.
Perforation, site of, in frontal sinus suppuration, 22.
Perforations in sigmoid groove, 17.
Perforator, Russian, for mastoid operations, 304.
Pericarditis, fœtid, 121.
Pericardium, affection of, in infective sinus thrombosis, 235.
Perineural sheaths as channels of infection, 52.
Periodic rigors in brain abscess, 132.
Periosteal elevator, 314.
Periostitis, acute, rare cause of pyæmic brain abscess, 121.
Periostitis, alveolo-dental, 246.
Periostitis of jaw cause of infective sinus thrombosis, 231.
Peripheral "soldering" of membranes, 80, 85.
Peritonitis, infective, 121.
Perivascular arterial sheaths as channels of infection, 51, 74.
Perivascular extravasations, 87, 102.
Perspiration in sigmoid thrombosis, 266.
Petro-occipital ligament, 33.
Petrosal sinus, inferior, 30 ; superior, 30.
Petrosal sinuses in relation to cavernous sinus, 29.
Petro-squamosal sinus, 8; persistence of, 32.
Petro-squamosal suture, at birth, 11.
erosion at, 72.
in relation to tegmen tympani, 4.
position of, 8.
route of infective extension, 80.
Petro-squamosal veins, thrombosis of, 68.
Petrous bone, ossification of, 2 ; description of, 13 ; caries of, 67.
Pharyngeal ulceration cause of cavernous sinus thrombosis, 246.

Phlebitis of sinuses, 231.
Phlebitis of veins in sigmoid thrombosis, 264.
Pia mater, 47 ; adhesion to thickened dura, 72.
nerve supply of, 48.
over area of cerebral degeneration, 107.
relation to cortex, 48.
relation to perivascular sheaths of vessels, 48.
vascularity of, 48.
veins and lymphatics of, 72.
Pneumonia, disintegrating, 121.
infective, in sinus thrombosis, 239.
Polyuria in brain abscess, 138.
Pons, abscess in, 122.
abscess in, symptoms, 195.
effects of pressure on, 137.
pressure on, in cerebellar abscess, 195.
Post-convulsive hemiplegia in frontal lobe abscess, 183.
Posterior-auricular lymphatic glands, 39.
Posterior cervical triangle, abscess in, 26, 234 ; abscess in, cases, 286, 287 ; pain on pressure in, 265.
Posterior condylar vein, 26 ; thrombosis of, 234.
Posterior meningeal artery, 33.
Posterior root of zygoma as surgical landmark, 9.
"Potts' puffy tumour," 61.
Pressure as cause of infective sinus thrombosis, 232.
influence of, on pulse and temperature, 114, 135.
in red softening, 102.
in white softening, 105.
of tumour cause of sigmoid thrombosis, 261.
Pressure symptoms in abscess of cerebellum, 113.
in brain abscess not proportional to size, 112, 114.
in large temporo-sphenoidal abscess, 113.
Probe, use of, in detecting erosions, 143.
Processes of dura mater, 42.
Prognosis in brain abscess, 223 ; in cavernous thrombosis, 251 ; in sigmoid thrombosis, 268.
Promontory, 3.
Prophylaxis of intracranial infective disease, 291.
Prostration in brain abscess, 132 ; in cerebellar abscess, 194 ; in infective sinus thrombosis, 240, 241.
Psychical blindness in brain abscess, 155.
Pterion, 147 ; as guide to Broca's lobe, 322.
Pterygoid plexus of veins, 33.
in relation to cavernous sinus, 29.
thrombosis, 246.
Ptosis in abscess of pons, 195.
in cavernous thrombosis, 247, 250.
in temporo-sphenoidal abscess, 154.
Puerperal state cause of marasmic thrombosis, 228.
Pulmonary affections cause of marasmic thrombosis, 228.
Pulmonary lesions, in infective sinus thrombosis, 235 ; in sigmoid thrombosis, 266.
Pulmonary type of infective sinus thrombosis, 238 ; symptoms of, 238.
Pulse, effect of evacuation of brain abscess upon, 114, 136.
Pulse, in acute meningitis and sigmoid thrombosis, 135, 136.
in brain abscess, 2nd stage, 135.
in cerebellar abscess, 194.
in cerebellar abscess in fourth ventricle, 195.
in infective sinus thrombosis, 237 ; influence of chloroform narcosis on, 237.
in intracranial hæmorrhage, 114.
in tubercular meningitis, 114.
in brain tumour, 114.
in brain abscess, 114.
Punctured fractures of skull and intracranial disease, 57.
Punctured wounds of skull and intracranial disease, 56.
Pupil, dilated, from intra-membranous injection of fluid, 43 ; in brain abscess, 145.
Pupil, in general cerebral meningitis, 146.
in cavernous sinus thrombosis, 146, 247, 250.
in cerebellar abscess, 195.
in frontal lobe abscess, 183.
in temporo-sphenoidal abscess, 154.
Pupils in pressure on pons, 195.
Purulent encephalitis, 102.
Purulent lepto-meningitis, 87.
Pus, exploring for, in brain abscess, 315.
from mastoid foramen, 26.
from middle ear in cerebral abscess, 132.
in cerebral abscess, character of, 106.
micro-organisms found in, 107.
otorrhœal, micro-organisms in, 53.
seen escaping from erosion in tegmen tympani, 143.
Pyæmia, result of intracranial infective lesions, 122.
Pyæmic abscess of brain, 121, 122 ; specially in occipital lobe, 123.
Pyramid, in relation to fossa subarcuata, 14 ; to facial canal, 15.

Rash in infective thrombosis, typhoid type, 240.
Recovery in sigmoid thrombosis even after systemic involvement, 269.
"Red softening" from marasmic thrombosis, 229.
Red softening in encephalitis, 102.
Reflexes in brain abscess, second stage, 141.
"Reflex method of propagation" of inflammatory particles, 51.
Reimplantation of bone disc, 314, 319.
Relations, supposed, of mastoid disease to cerebellar abscess, and of tympanic disease to cerebral abscess, 81.
Relief of pain on free escape of pus from ear, 132 ; in second stage of brain abscess, 132.
Remittent temperature in infective thrombosis, 237.
Removal of disintegrated thrombus from sigmoid sinus, 309.

Removal of ossicles, 302; effect of, on hearing, 302.
of sloughs from temporo-sphenoidal abscess, 316.
of suppurative foci in various situations, 292.
Repair after evacuation of brain abscess, 123.
Repeated free discharges of pus from ear, relation to cerebral abscess, 83.
Repeated rigor not a feature of abscess, but of infective thrombosis, 131, 132.
Respiration, arrest of, from pressure on pons, 137.
"Cheyne Stokes," 196.
in bursting of abscess into fourth ventricle, 195.
in cerebellar abscess, 137, 194.
in infective thrombosis, 138.
in lepto-meningitis, 137.
in purulent lepto-meningitis of cerebellar fossa, 133, 137.
in second stage of brain abscess, 137.
Respiratory difficulties during operation for cerebellar abscess, 321.
movement, effect on jugular veins, 34.
obstruction, cause of marasmic thrombosis, 228.
stoppage in cerebellar lesions, 196.
Retention of urine in brain abscess, 2nd stage, 138.
Retinal vessels, thrombosis of, 230.
Retraction of head in cerebellar abscess, 194.
in sigmoid thrombosis, 267.
Retro-pharyngeal abscess by extension from otitis, 84.
in relation to infective thrombosis, 231.
REVERDIN, case of cavernous sinus thrombosis from anthrax of face, 226.
Right intracranial invasion in relation to left, 82.
Rigidity of masseters in cerebellar abscess, 195.
of muscles in marasmic thrombosis, 230.
of sterno-mastoid muscle in sigmoid sinus thrombosis, 133.
Rigors following pulmonary infarctions, 266.
in brain abscess, initiatory stage, 131; periodic, 132; second stage, 140.
in infective sigmoid thrombosis, 262.
in infective thrombosis, 237; meningeal type, 241; typhoid type, 240.
in suppurative otitis media, 132.
Risks of leaving sloughs in brain abscess, 316.
RÖHRER, on non-pathogenic bacilli producing fœtor, 55.
Rotatory bur for mastoid operations, 299.
Rotatory nystagmus, 21.
Routes of access to brain for infective matters, ample, 56.
Rupture of brain abscess into ventricles or subdural space, 110, 111.
Rupture of brain abscess, 2nd stage, 144.

Santorini, veins of, 24.
Scalp wounds, in causation of infective thrombosis, 231.
method of rendering aseptic, 291.
SCHEDE, case of cerebral abscess, 326.
SCHWARTZE, indications for opening mastoid, 295.
Searcher for mastoid operations, 299.
Seats, most frequent, of brain abscess, 122.
Second stage of brain abscess, symptoms of, 132.
Semicircular canals, 21; effects of injury, 21.
Sensory aphasia in temporo-sphenoidal abscess, 154.
Sequence of paralysis, in temporo-sphenoidal abscess, 152.
Sigmoid groove, 16; knee of, 16; guiding surface lines, 16, 307, 308.
discoloration of, in infective thrombosis, 234.
erosion, 67, 72.
necrosis of, 69.
perforation of, 68.
relation to external auditory meatus, 16.
right and left, compared, 17; Körner's measurements, 17.
Sigmoid sinus, 24.
close relation to mastoid cells, 72; granulations from, 72.
exposure of, in operation for cerebellar abscess, 320.
in carcinoma of middle ear, 128.
in jugular foramen, 33; relation to jugular bulb, 36.
in tubercle of middle ear, 125.
operation to expose, 307.
position of, in relation to osseous meatus, 308.
position of, relative to supra-meatal triangle, 308.
process of thrombosis in, 73; disintegration of thrombus in, 73.
relation to antrum, 11; to mastoid cells, 13.
relation to cerebellum, 28.
removal of granulation tissue from, 320.
source of blood in right and left, 25.
Sigmoid sinus thrombosis, *vide* Infective sigmoid sinus thrombosis.
Sigmoid sinus thrombosis, 260.
ætiology, 260.
cases, 269-286.
diagnosis from brain abscess, 222.
symptoms, 262.
treatment, 307.
Sinus, cavernous, 29; relation to carotid artery and to nerves, 33.
Sinus, lateral, groove of, 17; guiding line, 17.
sigmoid, 24.
superior longitudinal, 24.
Sinus thrombosis, from pathogenic affections of face and scalp, 61.
may cause pyæmia, 122.
Sinuses, infective thrombosis, typhoid type, 240.
infective thrombosis, micro-organisms in, 53.
intracranial, thrombosis of, 226.
marasmic thrombosis of, 227.
mechanism preventing aspiration of, 33.
Size of brain abscess, 113.
Sloughs, in brain abscess, risks of and removal of, 317.

Sloughs, in cerebral abscess pus, 106, 115.
Slow pulse and high temperature, significance of, 135.
Softening in purulent encephalitis, red, 102; white, 105.
Softening of brain over thickened membranes, 80.
"Soldering" of brain membranes, 49.
"Soldering" of brain membranes over tegmen, 80.
Somnambulistic state in temporo-sphenoidal abscess, 154.
Source of gustatory fibres, chorda tympani, 20.
Special sinuses, thrombosis of, 242.
Speech in cerebellar abscess, 195.
Spheno-maxillary veins, 33.
Spheno-parietal sinus, 29.
Sphenoid, caries of, causing cavernous thrombosis, 246.
Spina bifida, 46.
Spinal lepto-meningitis, symptoms, 241.
Spinal membranes, extension of purulent lepto-meningitis to, 87.
Spinosum, foramen, 18.
Squamosal of temporal, ossification, 2.
in infancy, 8.
descending plate of, 11.
necrosis of, 84.
trephining through, for temporo-sphenoidal abscess, 313.
Squint in superior longitudinal sinus thrombosis, 243, *vide* Strabismus.
Stages of course of brain abscess, 130.
Staining of cerebellum in sigmoid infective thrombosis, 234.
Staining of extracranial tissues in infective thrombosis, 234.
Staphylococci in cerebral abscess pus, 106.
Staphylococcus pyogenes, albus, in intracranial pus, 54.
aureus, in intracranial pus, 53, 54.
citreus, in intracranial pus, 54.
Staphylococcus salivarius pyogenes (Biondi), in suppurating frontal sinus, 54.
Stapedius muscle, 6; nerve to, 19.
Stapes, 3; preservation of, at operation, 305.
Statistical table of, ablations of mastoid antrum, 329.
infective intracranial lesions, 332.
intracranial abscess, 333.
Sterno-mastoid muscle, rigidity of, in brain abscess with sigmoid thrombosis, 133.
Stimulation of chorda tympani, 20.
Stitches, localized, in chest in infective sinus thrombosis, 238.
Stools in infective thrombosis, typhoid type, 240.
Strabismus in cavernous thrombosis, 250.
in marasmic thrombosis, 230.
in infective thrombosis, meningeal type, 241.
Strength, diminution in ability to apply, in brain abscess, 134.
Streptococci in cerebral abscess, 106.
Streptococcus pyogenes, in intracranial pus, 53, 54.
Stupor in cerebral abscess, 143.
Styloid of temporal, ossification, 2.
Stylo-mastoid branch, posterior auricular artery, 5.
Subarachnoid fluid, effect of forced respiration on, 46; escape of, through nose, 47.
Subarachnoid space, 44; at base of brain, 44; "water bed," 44; over corpus callosum, 44; communications with ventricles, 44.
Subdural abscess, 50, 75; cases of, 75-79.
in middle fossa, 81.
Subdural space, 42; rupture of brain abscess into, 110, 111, 114.
Submastoid pain, in infective thrombosis, typhoid type, 240.
Suboccipital pain, in infective thrombosis, typhoid type, 240.
Subperiosteal mastoid abscess, 64; symptoms, 65.
Sudden onset in brain abscess, initiatory stage, 130.
Sulcus in base of temporo-sphenoidal lobe route of cerebral invasion, 80.
Superficial brain ulceration, 75; cases of, 75-79; in tubercle of middle ear, 125.
cerebral abscess, results of operations, 333; possible classification of, as true cerebral abscess, 125, 333.
petrosal nerves, 19.
Superior longitudinal sinus, 24.
longitudinal sinus thrombosis, 242; symptoms, 243; cases, 243, 244.
petrosal sinus thrombosis, 260.
semicircular canal, level of, 18; in infancy, 11; relation to tegmen tympani, 4.
vena cava, disintegrating thrombosis in, 236.
Suppuration, extradural, from injury of scalp without visible wound, case, 289.
in cerebellar fossa, abscess in neck from, case, 287.
in frontal sinuses, intracranial extension from, case, 289.
in frontal sinuses with perforation of orbits, cases, 287, 288.
in mastoid antrum, danger of, 293.
in temporo-maxillary joint from otitis, 84.
Supra-meatal triangle, 7, 297; boundaries of, 9; constancy of, 10.
Surface guides to position of sigmoid sinus, 307.
Surface, internal, of brain abscess, appearances, 106.
Sustained attention, want of, in brain abscess, 133.
Suture, petro-squamosal, 8, 11; masto-squamosal, 8, 11.
Swallowing of infective discharge, 266.
Swelling over mastoid in cerebral abscess, 143.
Symptoms, of abscess of brain, 130; second stage, 132.
of cavernous thrombosis, 247.
of cerebellar abscess, 194.
of empyema of frontal sinuses, 151.

Symptoms of frontal lobe abscess, 183.
of infective thrombosis mask brain abscess, 242.
of marasmic thrombosis, 229.
of meningitis may mask sigmoid thrombosis, 266.
of parietal lobe abscess, 193.
of pressure by abscess on middle lobe of cerebellum, 113.
of sigmoid thrombosis, 262.
of spinal lepto-meningitis in infective thrombosis, meningeal type, 241.
of thrombosis, generally mask meningitis and brain abscess, 267.
Systemic infection from sigmoid thrombosis, 266.
from sinus thrombosis, 235.

Tables, statistical, 329, 332, 333.
Tegmen antri, 4, 7.
Tegmen tympani, 4.
erosion of, 67.
escape of pus from, seen by auriscope, 143.
evacuation of brain abscess through, 313.
foramina in, 4; defects in, 4.
fracture of, 67.
injections through apertures in, 313.
in relation to mastoid antrum, 11.
necrosis of, 69.
pathological evacuation of abscess through, 82.
perforation, 68.
relation to abscess in middle fossa, 80.
scrutiny of, during operation, 300.
triangular plate of, 11.
Telæ choroideæ, 47; involvement of, in purulent lepto-meningitis, 87.
Temperature, in abscess bursting into fourth ventricle, 195.
in brain abscess, 114; initiatory stage, 132; second stage, 134; after evacuation, 135.
in cerebellar abscess, 195.
in idiopathic encephalitis, 114.
in infective peritonitis, 114.
in infective sigmoid thrombosis, 262; after lung involvement, 266.
in infective thrombosis, 237.
in meningeal type of infective thrombosis, 241.
Temporal bone, anatomy of, 1.
in child, in relation to otitis media, 64.
in relation to age, 11.
ossification of, 2.
relation of, to temporo-sphenoidal lobe, 17.
Temporal diploic veins, 30.
Temporo-maxillary joint, suppuration from otitis in, 84.
Temporo-maxillary vein, 6.
Temporo-sphenoidal abscess, aperture into, 81.
cases of, 155-182.
complicating infective thrombosis, 242.
from erosion of tegmen, 68.
localizing symptoms, 151.
of large size, pressure effects, 113.
operative treatment, 313.
pathological evacuation, case of, 110.
recurrent, 333.
Temporo-sphenoidal abscess, results of operations, 332.
size of, 113.
successful operations, 326.
with cerebellar abscess, cases, 122.
Temporo-spenoidal lobe, basal sulcus in, route of invasion, 80.
relation of, to temporal bone, 17.
thrombosis of arteries of, 72.
emboli in arteries of, 72.
total necrosis of, case, 115.
Tendency to doze in brain abscess, 2nd stage, 133.
Tenderness on percussion over mastoid and squamous in brain abscess, 133.
Terminal stage of cerebral abscess, symptoms, 143.
THIBAULT, case of cavernous sinus thrombosis, 226.
Thinness of tegmen, 4.
Thrombic barrier to general infection, 265.
Thrombic extension from veins in infective thrombosis, 231.
Thrombosed vessels, free ends in brain abscess cavity, 106.
Thrombosis, formative, 268.
in cerebral veins, effects of, 228.
in cerebral necrosis, 114.
in mastoid vein, 25.
infective, of sinuses, micro-organisms in, 53.
infective or inflammatory, 231.
marasmic, 227.
of arteries of temporo-sphenoidal lobe, 72.
of cavernous sinus, 245.
of cerebellar arteries, 74.
of cerebellar veins, 74.
of internal carotid, 51.
of internal jugular, in sigmoid sinus thrombosis, 265.
of intracranial sinuses, 226.
of petro-squamous veins, 68; of veins to sigmoid sinus, 68.
of posterior condylar vein, 234.
of pterygoid plexus, extending to cavernous sinus, 246.
of retinal vessels, 230.
of sigmoid sinus, 260.
of sigmoid sinus, process of, 73.
of special sinuses, 242.
of superior longitudinal sinus, 242.
of superior petrosal sinus, 260.
of veins, 51; of cortical arteries, 51.
of veins extending to sigmoid sinus, 261.
Thrombus, disintegration of, 232.
Tongue in brain abscess, initiatory stage, 132.
Tongue in infective thrombosis, 238; in typhoid type of, 140.
Tonic contractions in meningeal type of infective thrombosis, 241.
Tonsils, inflammation of, in relation to infective thrombosis, 231.
Tonsils, ulceration of, cause of cavernous sinus thrombosis, 246.
Torcular Herophili, 35.
Toxæmic irritation in sigmoid thrombosis, 267.

Transient aphasia in frontal abscess, 183; case of, 191.
Traumatic abscess of brain, 58.
Traumatic brain abscess, majority single, 122; location of, 122.
Traumatism as cause of infective thrombosis, 231.
Traumatism, brain "anchoring" and epilepsy from, 124.
Treatment, 291.
of wounds of dura and brain, 292.
of chronic otitis media, 293.
of abscess in Broca's lobe, 322.
of abscess in frontal lobe, 321.
of carcinoma of middle ear, 129.
of cerebellar abscess, 320.
of extradural abscess, 312.
of infective lepto-meningitis, 313.
of infective meningitis, 325.
of infective sigmoid thrombosis, 307.
of respiratory difficulties during operation for cerebellar abscess, 321.
of subdural abscess with ulceration of brain, 313,
of temporo-sphenoidal abscess, 313.
Tremors in marasmic thrombosis, 230.
Trephine, dangerous, in opening mastoid antrum, 305.
Trephining in acute serous lepto-meningitis, 86.
for temporo-sphenoidal abscess, 314.
in compound fractures of skull, 292.
Triangle, supra-meatal, 7, 9, 297.
Tributary veins as routes for distribution of products of disintegrating thrombosis, 232.
Tubercle of middle ear, 124; clinical course, 124-126; case of, 126; lepto-meningitis from, 125; ulceration of brain from, 125.
Tubercular caries of petrous bones, 67.
degenerating foci and pyogenic processes, 126.
meningitis, 67, 125.
meningitis, pulse in, 114.
otitis media, 84.
Tumour, cerebral, diagnosis from abscess, 223.
Tumour of brain, pulse in, 114.
Two forms of sinus thrombosis, 227.
Two modes of origin of infective thrombosis, 231.
Tympanal of temporal, ossification, 2.
Tympanic attic, 4.
branch of glosso-pharyngeal nerve, 19.
branch of internal maxillary artery, 5, 7.
ossicles, 5.
plexus of nerves, 19.
ring in infant, 3.
suppuration, optic neuritis in, 142.
Tympanum, 3.
at birth, 11.
communications with, 5, 6.
floor of, 4.
infective extension from, 67.
inflammation of, relation to intracranial suppuration, 62.
mucous membrane of, 4; in fœtus, 11; in adult, 12.
passage to antrum from, 4.
Tympanum, tegmen or roof of, 4.
Typhoid type of infective thrombosis, 240.
Typhoid type of thrombosis, especially in sigmoid, 266.

Ulceration of brain, 50, 75.
cases of, 75-79.
results of operations, 333.
treatment, 313.
Ulceration of sinus walls in infective thrombosis, 232.

Vascularity of lines of junction of different parts of temporal bone, 2.
of pia mater, 48.
Vein from cochlea to jugular bulb, 5.
temporo-maxillary, communicating with tympanum, 6.
Veins, diploic, 30.
distension, in cavernous sinus thrombosis, 250.
distension, in marasmic thrombosis, 230.
distension, in sigmoid thrombosis, 263.
emissary, 25.
of dura mater communicating with tympanum, 6.
of Galen, 23.
of Santorini, 24.
of Santorini in longitudinal sinus thrombosis, 242.
spreading thrombosis in, 51.
Velum interpositum, 47.
Venous intracranial circulation, 23.
Venous obstruction, cause of hæmorrhagic necrosis, 105.
in cavernous sinus thrombosis, 247.
in marasmic thrombosis, 230.
in sigmoid thrombosis, 262.
Venous plexus, basilar, 30.
Venous sinuses, intracranial, formation of, 41.
Venous supply of tympanum, 5.
Ventricles, distension of, in marasmic thrombosis, 229; lateral, over-distended, percussion note, 148.
VERNEUIL, cavernous sinus thrombosis from anthrax of face, 226.
Vertigo from injecting fluid into middle ear, 21; from perverted ocular or auditory impressions, 21.
from pressure on semicircular canals, 21.
in brain abscess, second stage, 138.
often accompanies vomiting, 21.
Vesalian vein, 29.
Vessels, pathogenic extension along, 50.
Vidian nerve, 19.
Villi, arachnoid, 45.
Virulency of otorrhœa, odour no test, 55.
VOLTOLINI, on inflammatory extension along fossa subarcuata, 14.
Vomiting and giddiness, significance of, 22.
Vomiting and headache, significance of, 138.
Vomiting in brain abscess, initiatory stage, 131.
in brain abscess, second stage, 138.
in infective thrombosis, 237.
in infective thrombosis, meningeal type, 241.

Vomiting in infective thrombosis, typhoid type, 240.
in simple otitis media, 132.
Von Dusch, on marasmic thrombosis, 226.

Wagner, cases of cerebral abscess containing oidium albicans, 122.
Wall, external, of antrum, 7.
Wall, internal, of antrum, depth of, 7.
Washing out brain abscess, method of, 317.
"Water bed" of subarachnoid fluid, 44.
White matter of brain, abscess in, 72.
White softening of brain, 105.
White swelling of neck, 39.
Wound, closure of, in operation for brain abscess, 319.
Wounds of dura mater and brain, treatment of, 291.
Wounds of orbits, treatment of, 293.
Wounds, punctured, penetrating bone of skull, 56.
Wounds, scalp, method of rendering aseptic, 291.

Yawning in cerebellar abscess, 195.

Zanfal, case of sigmoid thrombosis from acute catarrh of middle ear, 261.
Zenker, on cerebral abscess containing oidium albicans, 56.
Zygoma, posterior root of, as surface landmark, 9.

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